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URETERAL CALCULI—

AN UNUSUAL CASE WITH FIVE STONES IN ONE URETER*

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Ureteric stone in the aged is unusual and multiple ureteral calculi in elderly women are very rare. Huggins, of the Cleveland Clinic, in a study of 350 cases of stone in the ureter found the eldest female to be 67 years. Joly, in his work entitled Stone and Calculous Disease of the Urinary Organs, shows a photograph of six facetted calculi removed from a right ureter. The sex of the patient is not designated. He mentioned a person above 70 years of age who was operated upon for ureteral calculus at St. Peter's Hospital for Stone in England. Bumpus and Scholl found three cases above 70 years of age in a series of 880 cases of stone in the ureter treated at the Mayo Clinic. Herman, in his textbook on urology, presents a photograph of an x-ray film showing five stones in one ureter. The patient was operated upon at the Pennsylvania Hospital.

Your attention is called to this case because of the number of ureteral stones present, the patient's age, and the management employed.

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CASE REPORT

Mrs. W. P., aged 72 years, native and resident of Texas, decipara, widow, was first seen on May 19, 1944, at which time she complained of "blood and pus in the urine." The present history revealed that gross hematuria had been observed "off and on for about one month" during which time the urinary index was D 15-20/N 1-5. There had been relatively little pain although at times it manifested itself as "soreness in the bladder region" and burning upon micturition. At no time had opiates been required. Nausea and vomiting had occurred and five or six times the oral temperature reached 100° F. Chills had not been experienced. A loss of five pounds of weight transpired in the course of a year.

The past history disclosed that the first attack of "kidney and bladder trouble" was experienced in the summer of 1939. At that time pus was found in the urine and treated with urotropin for a period of "two or three weeks." She stated that x-ray films were not obtained then. The second episode took place in December, 1943, and was accompanied by fever, pyuria, and gross hematuria. Again, roentgenographic and cystoscopic studies are said to have been omitted. She received sulfathiazole orally for about one week. There have been no operations, injuries, or other known serious illnesses. Urinary stones have not passed.

According to the obstetric history there have been twelve pregnancies, of which ten reached term and resulted in the delivery of normal offspring. Two of the labors, the first and last, were rather long but were otherwise uncomplicated. Two pregnancies terminated spontaneously at about the eighth week without complications. The husband died of "heart disease." The menopause produced no difficulties and there has been no postmenopausal uterine bleeding or vaginal discharge.

Except for "kidney trouble" claiming the life of the patient's father, there is a negative family history for renal disorders, cancer, diabetes and tuberculosis. This woman has always enjoyed an adequate diet and her living conditions have been excellent. Coffee and tea have been imbibed con-

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servatively, and tobacco and alcohol have not been used.

Examination revealed a well developed female, of the stated age, 4 feet, 11 inches in height, and weighing 98½ pounds. Temperature, pulse, and respiration were normal. The blood pressure was 140/70 in both arms. Generalized arteriosclerosis was moderate. Arci senili were present. The teeth were spurious. Examination of the chest showed a slight enlargement of the heart. There was slight evidence of emphysema. The mammae were atrophic. The liver was slightly palpable, but the spleen could not be palpated. There was no evidence of nephroptosis or renal enlargement. Slight suprapubic tenderness was demonstrable. The perineum was relaxed and lacerated. Moderate asymptomatic cystocele and rectocele were present. Senile vaginal changes had occurred. Inspection of the cervix uteri and palpation of the uterus and other pelvic contents disclosed no pathologic findings. Slight asymptomatic external hemorrhoids were visible. Rectal examination showed no pathology. The extremities were free of edema and freely movable. Reflexes were psysiologic. Microscopic study of catheterized specimen of urine showed innumerable erythrocytes and many leukocytes. Hospitalization for complete investigation was advised; consequently, the next night (May 20) the patient was admitted to the Southern Baptist Hospital. The urine proved to be acid and contained 1+ albumin with a specific gravity of 1.009. The hemogram was as follows: red blood cells 3,510,000, hemoglobin 67 per cent, white blood cells, 8,100, S.M. 35, L.M. 2, N. 62, and E. 1. The platelets were normal. Bleeding time was 1 minute. Coagulation time was 4 minutes 30 seconds. Blood chemical findings were: Total N.N. 33.3, urea N. 16.6, creatinine 1.5, uric acid 3.27, and sugar 75. Blood Wassermann, Kahn, and Kline tests were negative. Blood sedimentation rate was 23 mm. in 60 minutes, presenting a diagonal line. An x-ray film was reported as follows: "View of the chest shows the transverse diameter of chest to be 24 cm. and that of the heart 12.5 cm. Lungs show emphysema, but are otherwise clear."

On May 23, the urethra was dilated with sounds number 18, 20, 22, and 24 (Fr.) in order to introduce a Brown-Buerger cystoscope. There was marked injection of the blood vessels of the bladder compatible with generalized acute and subacute cystitis. A small diverticulum was found and its interior easily inspected. The urine was cloudy but not grossly bloody. There were no ulcers, stones, or new growths. The bladder capacity was good. The ureteral orifices revealed no pathology. A number 6 catheter was easily introduced into each renal pelvis. Urinary stasis was minimal on both sides. Specimen collected through the right catheter was bloody and that on the opposite side was hazy. Indigo carmine injected intravenously appeared in the right in 4 minutes and concentrated excellently in 6 minutes, while the figures for the left kidney were 6.5 minutes for appearance and fair concentration in 10 minutes. KUB roentgenogram with radiographic catheters in place showed five shadows in the region of the right kidney and two in the left renal region, compatible with bilateral nephrolithiasis (see fig. 1).

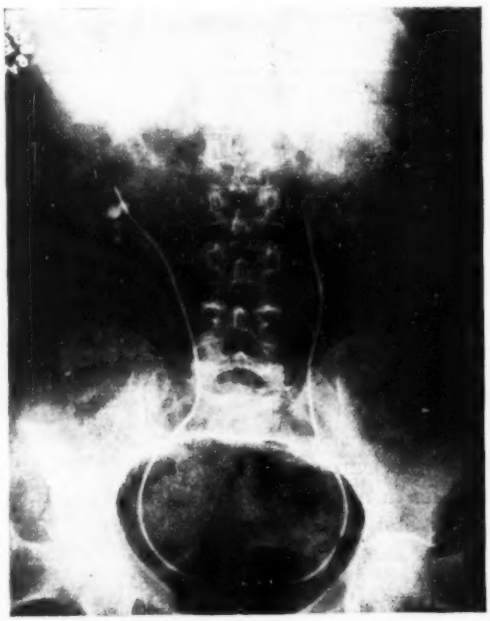

Fig. 1. KUB x-ray film with ureteral catheters in place revealed five calculi in the right renal pelvis. (May 23, 1944.)

Bilateral two-position pyelograms, using Skiodan as the solution for visualization, presented a picture of renal damage, worse on the left. Bacteriologic studies of the urine obtained at time of cystoscopy showed: Bladder—many pus cells, moderate number red blood cells, Gram negative bacilli, Gram negative cocci, and Gram positive cocci in chains; right kidney—many pus cells and red blood cells, Gram negative pleomorphic bacilli, and Gram negative cocci in chains; and left kidney—moderate number of pus cells and red blood cells, Gram negative and positive cocci, and Gram negative bacilli. Cultures proved the bacilli to be *Esch. coli*. Because of her renal status and age conservatism was advised; therefore, the patient was discharged on May 25, with a diagnosis of generalized hemorrhagic cystitis, small urinary vesical diverticulum, bilateral nephrolithiasis, chronic pyelonephritis, and generalized arteriosclerosis. Mandelamine tablets, three times a day, p.c., were prescribed in addition to Upjohn's ferrated liver concentrate with vitamin B complex, two capsules, three times a day, and Squibb's spe-

cial formula vitamin capsules, one twice a day. High fluid intake was advised.

During the next month the patient was seen at the office for bladder irrigations and advice as to medication. After the course of mandelamine had been completed sulfadiazine was administered and subsequently discontinued because of nausea. Likewise sulfathiazole could not be tolerated; consequently, neo-prontosil was tried and proved satisfactory. The weight increased six pounds and the patient was "feeling O.K." until June 23, at which time severe pain occurred in the right renal region requiring the administration of dilaudid. Four days later the pain recurred to an excruciating degree. Again dilaudid was given intramuscularly and the patient returned to the Southern Baptist Hospital June 27, 1944. She was still afebrile and had had no chills. A skiagram revealed the presence of five stones in the middle third of the right ureter, all of which had been in the kidney pelvis at the time of first hospital admission (see fig. 2). Because of the location and number

Fig. 2. Roentgenogram obtained June 28, 1944, showed five stones in the middle third of the right ureter.

of stones, and the very poor condition of the left kidney, and in spite of the patient's age, surgical removal of the calculi was advised. However, the relatives were firm in their belief that operation would prove fatal and the patient insisted that she would "pass those stones if given a little help with a cystoscope." So, on June 29, ureteral bougies no. 4, 5, 6, 8, 10, and 11 were passed into

Fig. 3. KUB x-ray film with cystoscope and ureteral bougie in place disclosed displacement of the stones (June 29, 1944).

Fig. 4. Skiagram obtained July 3, 1944, proved downward progress of three calculi.

the right renal pelvis. Figure 3 shows the displacement of the stones with the largest bougie in place. Before the patient was removed from the cystoscopic room the bougie was withdrawn and a no. 5 and no. 6 catheter left indwelling. Incidentally, intravenously injected indigo carmine did not appear in 10 minutes. Forty-eight hours later the catheters were removed and two days thereafter her temperature reached 102°. Dilaudid hydrochloride gr. 1/32 was given by hypodermic injection as needed for pain, and fluids were forced. One octin mucate tablet, grains 2, was prescribed three times a day, beginning July 1. KUB x-ray film obtained July 3 proved that three stones had progressed downward, as shown by figure 4. Being free of fever and wishing to stay with relatives on July 4, the patient was permitted to leave the hospital. The vitamin, iron and liver, and neo-prontosil administration was continued. Furthermore, a tablet of octin was advised three times a day and one of dilaudid as needed for severe pain. On July 6 a daughter telephoned to state

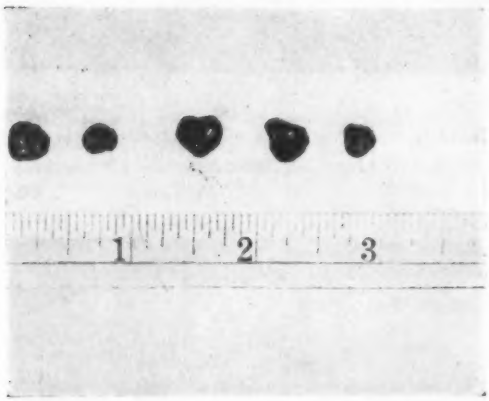

Fig. 5. Stone number 1 was passed via urethram on July 6, 1944; numbers 2 and 3 passed July 7, and the remaining two were passed on July 13.

that pain was marked and the administration of octin was advised every four to six hours in addition to dilaudid as needed. On that night a stone was passed. The next day two more stones were passed. On July 13 the remaining two calculi on the right were passed (see fig. 5). An x-ray film (see fig. 6) proved the right kidney and ureter to be free from stones, all of which were of the calcium oxalate variety.

Therafter the patient was seen several times. She improved remarkably and was "feeling fine" when she returned to Texas in September, 1944. A recent letter reports that complications have not been encountered.

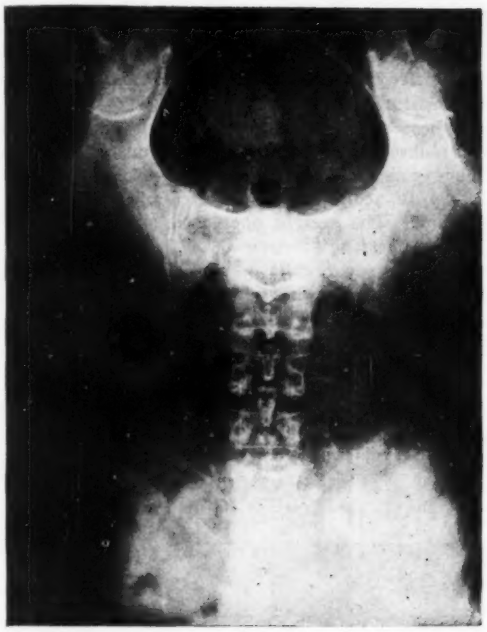

Fig. 6. Roentgenographic studies on July 15, 1944, proved the right ureter and kidney to be free from opaque calculi.

SUMMARY

The case of a 72 year old white female presenting five calculi in the right ureter was successfully managed without operation. Cystoscopic ureteral dilatation and the administration of an antispasmodic, octin, were employed.

Acknowledgment: Octin, *methyliso-octenylamine*, is an unsaturated aliphatic amine with antispasmodic properties. It was made available for use in this case through the courtesy of Mr. Drew Baker, of the Bilhuber-Knoll Corporation.

A CASE OF PERIRENAL ABSCESS*

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The following is the report of case of polycystic kidneys with a right sided perirenal abscess.

CASE REPORT

This patient, Mrs. L. R. P., aged 48, was admitted to Southern Baptist Hospital on December 20, 1944 and discharged January 1, 1945. She had been sick one week before admission to the hospital, complaining of pain in the right side,

*Read before the Orleans Parish Medical Society, February 12, 1945.

chills and fever. The patient had discovered a tender mass in the right lower quadrant. This mass extended upward towards the region of the right kidney. For the past six years she was known to have had hypertension and on admission the blood pressure was found to be 182 systolic and 100 diastolic.

She was a well nourished, strongly built woman and is the mother of five living healthy children. From time to time she has been treated for pyelitis, the most recent attack occurred about one month before admission to the hospital.

On admission her temperature was 101, pulse rate 112, respiration 24. The temperature reached 105° on the third day of her stay in the hospital. The day after admission she was cystoscoped by Dr. H. W. E. Walther and a pyelogram made. The urine from both kidneys was loaded with pus and bacteria and a culture showed *Escherichia coli.* The x-ray showed a filling defect of the lower pole of the right kidney, suggestive of a growth. This filling defect not only compressed the pelvis of the kidney but also compressed the lower calices. It was noted in the x-ray examination there was no scoliosis, although scoliosis is a fairly frequent finding in perirenal abscess.

On December 13, the right perirenal space was entered and a large quantity of foul pus was evacuated. It was noted that approximately 20 cysts projected from the cortex of the right kidney and these cysts ranged in size from 1 to 1½ cm. in diameter. Apparently one of the cysts at the lower pole had become infected, since one was found ruptured in the region communicating with the abscess and the perirenal abscess had resulted. All of the cysts which were visualized were punctured. A cigarette drain was placed at the lower pole of the kidney after 10 mg. of sulfanilamide had been put in the wound.

It was later necessary to reopen the wound and place another cigarette drain at the upper pole of the kidney; following this the patient made a stormy and slow recovery.

She was allowed to go home to the country on January 1. She developed intense pain in the left kidney; however, repeated lavage of both kidneys with the use of sulfonamides seem to have overcome the attack of pyelitis.

COMMENT

The unusual feature of this case is that the patient has had a number of children without any sign of failure of kidney function. Incidentally the P. S. P. for two hours was only 10 per cent. An older brother was operated on by me, 18 months before I saw this patient, for enormous polycystic kidneys. He has recovered sufficiently to return to his normal duties as driver of a school bus.

The ultimate prognosis in this case is poor.

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A SIMPLE TREATMENT FOR SEBACEOUS CYST*

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NEW ORLEANS

Some years ago I had occasion to remove a sebaceous cyst 2 1/2 cm. in diameter from the face of a rather attractive woman. It was situated just above the lower border of the right mandible. I made a low incision partly so that the resulting scar would not show so much and partly to avoid the possibility of injuring the lower branches of the facial nerve. I was so careful in avoiding nerves that the cyst ruptured in removal and I must have left a small piece of the cyst wall behind, for it promptly recurred. What to do then, was the problem. I felt that the dissection difficulties and danger to nerve branches would be greater now than before. So I began to wonder if there was not something else I could do besides excision. Then, one day, I remembered having heard the late Dr. Walter E. Sistrunk describe a procedure for the excision of unsightly skin scars of wide extent by removing a narrow strip in the center, suturing the edges of the resulting wound and after thorough healing, repeating the procedure as many times as was necessary finally to remove the entire area. It seemed to me that if I could excise or destroy a small area of the top of the cyst, let it heal and repeat the process till the entire lesion had been destroyed, the cyst could thus be finally made to disappear without danger to surrounding nerve branches.

Meanwhile the new cyst continued to grow. It finally occurred to me that by

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using the diathermy current through a needle inserted in the center of the top of the cyst a slough could be produced which would heal by cicatrization, making the cyst wall smaller and that the process could be repeated till the entire wall had been destroyed. With the patient's consent I proceeded to carry out this idea. I used the office diathermy machine, inserted the needle in the top of the cyst and produced a slough about 1/2 cm. in diameter. In due course of time the slough separated, leaving the cavity open and draining, and in a few weeks the cyst seemed to shrink and melt away as it were and pretty soon the floor of the cyst wall could be seen to present nearly level with the skin surface. There was no further secretion and what finally resulted was a depression resembling a wide mouthed recently emptied blackhead. Cosmetically the result was very much better than I could possibly have hoped for, in fact, it was hardly noticeable even on close inspection. I wondered at this result for a long time before I decided deliberately to try it a second time, and the result was again so good that I have now used it on a total of twenty patients, nearly all within the past two years. The size of the cysts has ranged from 1 1/2 cm. to 6 cm. in diameter. Four were inflamed and bordering on rupture at the time of treatment. They were located as follows: On the face, four; scalp, one; collar line of neck, two; breast, two; above the breast, one; below the breast, one; between the breasts, one; on back, six; deltoid region, one; high thigh amputation stump, one. In all cases the contents finally were extruded and the cyst wall and cavity grew smaller and smaller till the bottom of the cyst wall presented at the opening and in all but two cases levelled off with the surrounding skin, leaving nothing but a slightly pigmented spot to show where the opening had been. There was no tendency to cicatrical closure of the opening, though there was an average of about 50 per cent final narrowing. This area now lined with cyst wall epithelium eventually takes on all the qualities of skin. In the first case, and in the one situated in the scalp, the final result was a narrow but not very deep depression in the skin that looked like a blackhead that had just been emptied of its contents (fig. 2 j).

TECHNIC

A sharp needle is inserted into the center of the top of the cyst so that its point barely protrudes into the cavity (fig. 1)

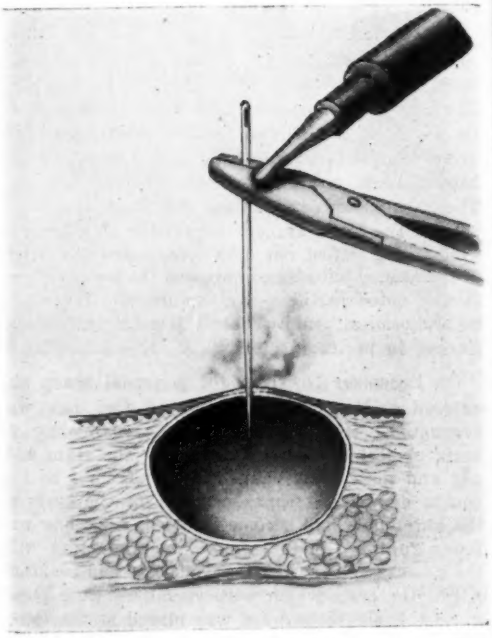

Fig. 1. Insertion of the needle and application of the electrode as the current is turned on.

and the current is turned on until a whitish eschar is seen to form around the needle. If the needle is inserted too deeply the force of the current is dissipated in the cyst contents and may give rise to steam formation if enough current is used to get desiccation or cauterization of skin and cyst wall. The diameter of the slough produced should be approximately one-fourth or one-third that of the cyst itself. One puncture is sufficient with cysts up to about 3 cm. in diameter. Two or three adjacent punctures will be required with the larger cysts. The center of the top of the cyst is the preferred point of puncture, for here the combined thickness of skin and cyst wall is thinnest,

there being no fat or areolar tissue intervening. A puncture away from the center would result in a much thicker slough (fig. 3a) with the possibility that cicatrical closure might ensue before skin and cyst wall epithelium had time completely to cover the entire edge of the resulting opening. Where the cyst has become infected and possibly ruptured into the surrounding fat, it may be difficult to judge just where the thinnest area over the cyst proper actually lies. In one case the needle was inserted into an adjacent pus cavity and the opening promptly closed, necessitating a second treatment with the needle properly placed. The unipolar desiccating current was used for the smaller lesions and the bipolar coagulating current for the larger ones. The current is barely strong enough to produce the necessary desiccation or necrosis, the patient experiencing a sharp stinging sensation that is easily borne. In only two nervous individuals was it neces-

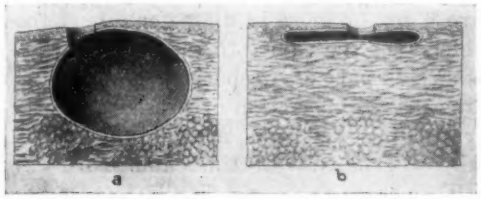

Fig. 3. (a) Increased thickness of resulting opening where needle has not been inserted in center of top of cyst; (b) persistence of cavity in case in which hard mass of inspissated cells had to be forcibly squeezed out before cavity diminished in size and finally closed.

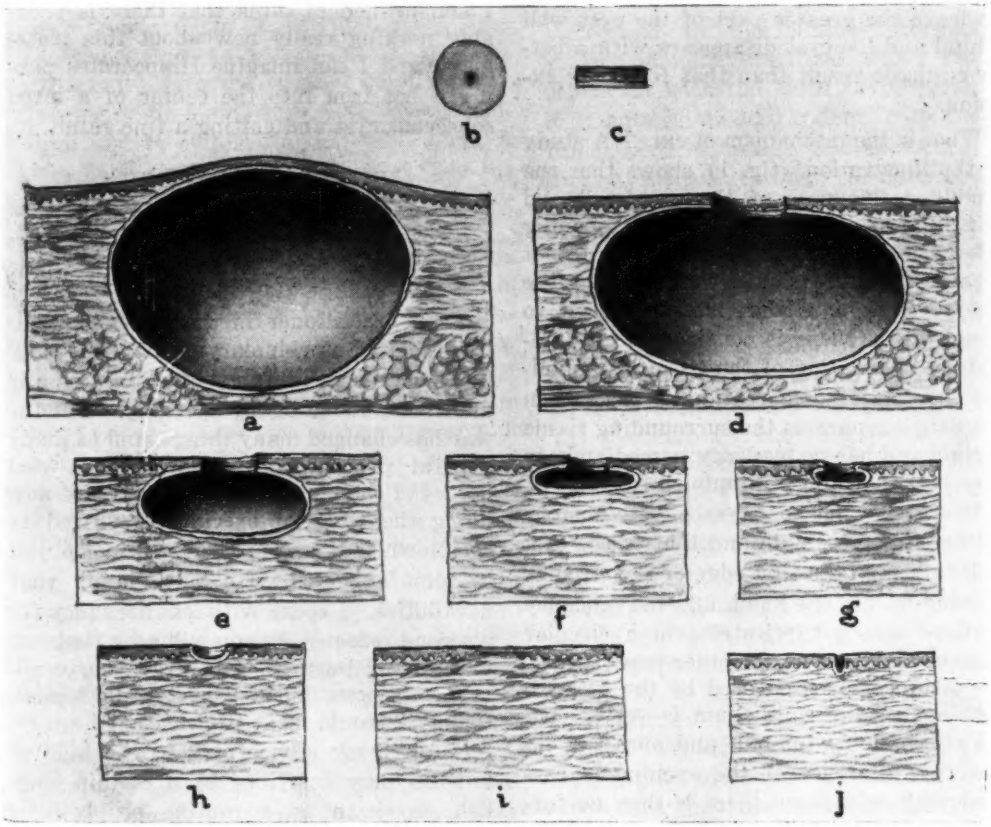

Fig. 2. (a) Section of intact cyst; (b) superficial view of button of tissue which sloughs out; (c) lateral view of b; (d) cyst after slough has separated; (e, f, g, h) various stages of diminution in size of cavity; (i) final levelling off of cyst wall, the result in 18 cases; (j) the result in two cases.

sary to use a 1 per cent procaine injection in the surrounding skin.

In the one in the deltoid region (fig. 2b) there was for some time no sign of diminution in size of the cavity. It was found that the contents consisted of a solid mass of inspissated epithelial cells which had to be squeezed out forcibly, following which the cavity promptly diminished in size and finally closed.

I had been taught that a sebaceous cyst must be removed whole and intact and that recurrence would ensue after any type of drainage, or if even the smallest portion of its wall remained behind. I have heard of failure to recur following infection so severe as to result in complete destruction of the cyst wall, but have never had this experience. Yet here, after one treatment, we leave the greater part of the cyst wall behind and the cyst disappears with a better cosmetic result than that following excision.

What is the mechanism of cure? A study of the illustration (fig. 1) shows that the combined thickness of the cyst wall and skin in the edges of the resulting opening after a disk of tissue sloughs away is not great. In fact, it is quite thin. During the five to eight days that it takes for it to slough away there is considerable inflammatory infiltration of the tissue surrounding the line of demarcation so that when the slough separates the surrounding tissue is rigid and has no tendency immediately to permit the opening to diminish in size.

There now ensues a race between the epithelium of the skin and that of the cyst wall to bridge the thin edge of the circular opening on the one hand, and the tendency of the opening to cicatrize in a circular manner and close on the other hand. Since the distance to be covered by the two advancing lines of epithelium is very short, the epithelium wins out, and once the entire circumference of the opening is covered with epithelium there is then no further tendency to cicatrical closure of the opening. Meanwhile, the contents of the cyst are free to drain out and actually do so. But what is most remarkable is the fact that the cyst cavity gets smaller and smaller till the bottom of the cavity finally presents on the surface and straightens out in a straight line (fig. 2 a, e, f, g, h, i). How this is brought about I find it hard to understand for some of these cavities were 6 cm. or more in diameter. Is it the tendency of the cavity to resume its original size as part of a sebaceous gland, or is it the massaging action of pressure on the surrounding fat and areolar tissue, or both? Perhaps with time and more experience these facts will finally be cleared. But in the meantime we are getting the results. I feel that the cautery effect of the treatment is essential to producing the desired result and that simple knife excision of a button of tissue would not be so successful. I have not tried simple excision. I am inclined to think that there is probably nothing really new about this treatment, and I can imagine Hippocrates poking a hot iron into the center of a large sebaceous cyst and getting a fine result.

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SINUS DISEASE IN CHILDHOOD*

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As a practitioner in the highly specialized field of otorhinolaryngology I usually feel a degree of hesitancy in addressing any audience outside of my specialty. But the war has changed many things, and so many general practitioners, who are the real heroes of the home medical fronts, are now doing the work of specialists as well as their own that perhaps you will not object to some suggestions to help you in your new duties. I speak with less hesitancy for a second reason. As you will soon find out, part of my purpose is to urge you to advise not more, but fewer, tonsillectomies, though I should make it clear that I am referring in my advice only to the kind of tonsillectomy described by a certain English physician as a routine prophylactic

*Read before the Iberville Parish Medical Society on February 24, 1945 by invitation.

†From the Department of Otolaryngology of the School of Medicine of Louisiana State University.

ritual, performed for no particular reason and achieving no particular result.

In 1918 Dean and Armstrong[1] read before the twenty-fifth annual meeting of the American Laryngological, Rhinological and Otological Society a communication which laid the groundwork of the modern concept of nasal and paranasal sinus disease in infants and young children. Much that is in it cannot be accepted today. The bacteriologic data need revision in the light of modern knowledge. The statement that 80 per cent of all cases of sinus disease in children can be cured by tonsillectomy has been thoroughly disproved. The frequency of a cause-and-effect relationship between sinus disease and arthritis would now be looked upon with a good deal of skepticism. Indeed, Dean[2, 3] himself retreated later from some of his original views. Yet he and his co-author, in spite of these criticisms, were the first to point out the frequency of sinus disease in young children and to emphasize that the reason it was not being diagnosed was because the possibility of its occurrence was not generally realized.

There is still no very general realization of the possibility. Piness and Miller[4], in discussing the allergic phase of sinus disease in children, wrote in 1939 that they found themselves obliged to say all over again the same things they had said in 1925 because there was still an almost universal lack of appreciation of the frequency and consequences of sinus disease in children.

The true incidence, it must be granted, is unknown. That it is as frequent as some enthusiasts claim, or as infrequent as some skeptics assert, is equally unlikely.

The only real scientific study is by Ebbs,[5] who found purulent infections of one or more sinuses in almost a third of 495 postmortem examinations on children under 14 years of age dying of a variety of medical and surgical conditions. Generally speaking, the incidence is greater in children in poor economic circumstances, though the disparity between them and children in more favored circumstances is less than might be imagined. Boys are more likely to be affected than girls, because they are more careless about protective clothing, habits of play, and antecedent upper respiratory infections. The incidence is greater in the winter months, especially in late winter when the resistance of children is likely to be at a low level and when the virulence of infecting organisms has been enhanced by human passage. The incidence in summer months has increased, however, due to a wider use of swimming pools, without proper precautions.

ANATOMY AND PHYSIOLOGY

One reason why sinus disease is not regarded as a more frequent possibility in young children is that the regional anatomy at that age is often forgotten and the nasal physiology is likewise overlooked.

Reduced to the simplest terms, the sinuses may be described as air cavities connected with the nasal cavity, and, like it, lined with ciliated columnar epithelium. It has been estimated that at least 10,000 liters of air pass through the nostrils every 24 hours, that at the lowest estimate 1,500 organisms are inhaled every hour, and that it must be a common event for 14,000 organisms to enter the nasal passages during an hour's tranquil respiration. The defense mechanism of the nose and paranasal sinuses, in addition to the phagocytic activity of certain blood cells, depends upon: (1) the coating of mucus in the nose, which serves as a protective blanket against these organisms, and (2) the ciliary activity of the mucosa, which serves as the motive power to keep the mucus moving toward the ostia of the sinuses and thence to the nasopharynx, carrying with it bacteria, cells and foreign particles. When either of these mechanisms is disturbed, sinus disease may be the end-result.

It is true that the sinuses, like certain other bodily structures, are underdeveloped in infants and young children, but it is not true that they are therefore incapable of becoming infected. The nasal sinuses, although they are little more than mucosal folds and recesses at birth, are usually well established by the third or fourth year.

The ethmoidal cells are present at birth, are well developed at two years of age, and can retain infectious material much earlier. The maxillary sinuses have been identified in a three months old fetus and are well developed at two years of age. Precocious development is by no means uncommon. Another fact of pathologic and diagnostic importance is that the sinus walls early in life consist of soft, cancellous bones.

Infection of the nasal sinuses during the period of development, if it is not corrected, definitely retards their normal growth and, in the case of the maxillary sinus, may interfere with dentition. Furthermore, infection of a single sinus may have a deleterious effect on another sinus. Shea[6] reported a series of cases in which development of the frontal sinuses was arrested because of infection in the antra and ethmoids; when the infection was corrected, normal growth was resumed.

ETIOLOGY

The etiology of sinus disease in children is both similar and dissimilar to its etiology at other periods of life. A large proportion of cases follow colds. Many cases follow exanthematous diseases. A thickened or deviated nasal septum, with or without a high palatine arch, is a frequent anatomic cause. Enlarged adenoids may act as a barrier both to the passage of air through the nose and to the passage of secretions from it, and, if they become infected, may themselves serve as a source of sinus infections. Basic causes include bad feeding habits; inadequate clothing; dry, overheated buildings; climatic conditions; unwise nasal medication; such constitutional factors as hypothyroidism; and poor hygiene in any respect. The theory that certain vitamin deficiencies predispose to changes in the epithelium of the sinuses and to subsequent infection has never been corroborated in human subjects, though vitamin imbalance probably contributes to a general lack of resistance.

Less familiar than these causes, and perhaps less readily acceptable because it is new, is the concept of sinus disease in childhood on an allergic background. Piness and Miller[4], in a remarkably good article on the subject, point out that allergy may be caused by a wide variety of agents and does not necessarily take the form of hay fever or allergy. Semenov[7], who made a histopathologic study of the mucosa in 500 cases of sinus disease, estimated the total probable incidence of allergic cases as 52.4 per cent. Dean,[3] who first implicated dietary habits in the etiology of sinus disease in children, later attributed it to a specific allergic diathesis. If one remembers that allergy is best defined as an individual hypersensitiveness, much of the confusion which attends all discussions of the subject would be promptly cleared away and the idea would cease to seem in any way unreasonable.

PATHOLOGIC CHANGES

The etiologic classification of sinus disease in children leads logically to its pathologic classification as: (1) purulent and allergic, or (2) as intumescent, catarrhal or purulent. The terms carry their own definitions.

The most prominent manifestation of any allergic condition is edema. In allergic sinus disease the pathologic chain of events consists of swelling of the turbinates; defective drainage; stasis; superimposed infections; finally ethmoiditis, possibly pansinusitis and, if repeated attacks occur, chronic sinus disease, often associated with persistent swelling of the turbinates, especially of the middle turbinates. The superimposed infection is frequently so dominant in late cases that the original allergic process is overshadowed.

In the non-allergic variety of sinus disease, as well as in the allergic variety in which secondary infection is present, the reaction in the tissues is sometimes more violent in children than in adults because it occurs in what may be called virgin territory. The epithelium in children is more fragile than in adults. The vessel walls are more delicate and therefore more permeable. The tunica propria is less fibrous and the structural cells less mature. As a result, the tissues are highly vulnerable to irritation and bacterial invasion, and the

reaction is intense because of the facility with which transudates or exudates become established in such circumstances. As the mucosa becomes engorged and edematous, so that the opposing nasal structures are almost or actually in contact, the cilia become inactive and, as the infection progresses, the sinuses are unable to empty because of continued engorgement of the mucosa at the ostia. The histologic picture is chiefly edema of the epithelium and subepithelium. The process of extension and repair is the same as in any similar lesion, modified because of local anatomy.

Unless secondary infection is superimposed, bacteriologic studies are not useful in allergic sinusitis. In the pyogenic variety any of the usual types of organisms may be found alone or in combination. The retained secretion is a favorable culture medium for organisms of low pathogenicity.

The maxillary sinus is chiefly affected in infancy. Since there is no real ethmoid sinus during the first two or three years of life, ethmoid involvement at this period is for all practical purposes an osteomyelitis. Anatomic considerations explain the frequency of orbital complications of the latter type of infection in early childhood.

CLINICAL PICTURE

The most frequent symptoms and signs of sinus disease in children, as in adults, are nasal and postnasal discharges, rhinitis, coughs, hoarseness, headache, local discomfort, and otitis media. Mouth breathing may be either a cause or an effect. General symptoms include failure to gain weight, irritability, restlessness, listlessness, malaise, anorexia and vomiting; many symptoms in this group are probably toxic manifestations of absorption from the foci of infection. While nephritis, pyelitis, chorea, arthritis and rheumatic fever are sometimes due to sinus disease, there should be due caution in attributing them to possible focal infection until every other cause has been excluded and the cause-and-effect relationship has been indubitably proved.

Sinus disease of allergic origin may be manifested by chronic nasal irritation without sneezing; frequent rhinorrhea; persistent sore throat without tonsillitis; "stuffy" ears without pharyngitis; chronic cough without bronchial infection, wheezing or dyspnea; and recurrent bronchopneumonia without identification of pneumococci. In allergic sinusitis there is almost always a magnification of symptoms which arise from acute blockage of the sinuses. If secondary infection occurs, the clinical picture does not differ from that of non-allergic sinusitis.

Headache is a difficult symptom to evaluate in young children. Older children with sinus disease complain of it in various areas. It is undoubtedly just as frequent in infants and young children, who merely cannot complain of it. It is variously due to blockage of the sinuses with increase of pressure, to toxic irritation, and to actual inflammation of, or pressure upon, the regional nerve supply.

Loss of the senses of taste and smell occurs in children with sinus disease, just as in adults, and is far more important than is generally realized. The olfactory area is located in the narrowest part of the nasal cavity, and the olfactory function is therefore dulled by the least turgescence or edema of the superior and middle turbinates. An adult who loses his sense of smell and taste in the course of an illness can make himself eat, but a young child cannot. Therefore a child who cannot be roused to interest in his food can often be profitably studied from the standpoint of possible sinus disease.

COMPLICATIONS

Local complications of sinus disease are due to direct extension of the infection into adjacent areas through the peripheral bony plate of the sinuses. The ethmoid cells are the most common pathways. Their walls are extremely thin and may be absent, so that the periosteum is brought into direct contact with the dura or, laterally, with the orbit. Infection therefore readily spreads to the meninges or the orbital cavity. Evidently, however, these are not common anatomic anomalies, or complications would be more frequent. Osteomye-

litis may give rise to the same spread of infection.

Whether bronchiestasis is a complication of sinus disease or merely occurs in accidental association with it is still to be determined. It is usually stated that the lymphatic drainage of the sinuses is to the cervical lymph nodes and thence to the thoracic nodes, but it has been shown experimentaly that it does not reach the lungs. Whether the association is causative or accidental, it is undoubtedly frequent and the treatment of bronchiectasis naturally involves the treatment of the sinus diseases.

DIAGNOSIS

Certain difficulties attend the diagnosis of sinus disease in children. The first is inherent in their age. Even the simplest examination may be difficult, and instrumentation, which is essential in all cases of sinusitis, may be very difficult indeed. If patience and tact do not achieve results, light anesthesia is necessary.

The second difficulty is that transillumination may seem negative when really it is not, because of lessened bone density at this age and the fact that lesions are fresh. As a result, the secretion is mucous instead of purulent, hypertrophies and polypi are infrequent, and changes which would show up in an examination of adults do not show up in children. In this same connection the warning should be issued that radiologic findings which are normal, when interpreted in relation to the age of the child should not be mistaken for pathologic changes.

Finally, if the patient is not seen until the disease is advanced, a number of confusing situations may arise. Disease of allergic origin may be obscured by a superimposed infection. A sinus which represents the major involvement when the patient is examined may not be the point of origin. Indeed, remote complications may entirely overshadow the local causative condition.

The diagnostic procedure should follow the same lines as in the adult, that is, a complete history; a complete physical examination; anterior rhinoscopy; nasopharyngoscopy; transillumination; roentgenologic examination; and cytologic and bacteriologic studies as indicated. Topical or general anesthesia should be used as necessary if a complete examination is not otherwise possible.

Cases in which an allergic origin is suspected should be investigated by the standard routine, with such individual studies as the circumstances may indicate. The history in such cases frequently furnishes the clue to the etiology, as do certain special findings. Polypoid degeneration of the mucosa is an almost invariable mark of allergy, as is enlargement of the tonsils without evidence of infection. Re-growth of lymphoid tissue after tonsillectomy and recurrence of adenoids, according to Piness and Miller,[4] are both extremely important. These authors observed lymphoid hyperplasia of the tonsils in 80 per cent of 95 allergic children and recurrence of tonsillar tissue in 544 tonsillectomized children. The recurrence in some instances was observed within two months of operation, and in six cases was great enough to warrant a second tonsillectomy. They very correctly stress the importance of accurate diagnosis in such cases, to increase the probability of therapeutic relief, to decrease the probability of misdirected therapeutic effort, and to prevent secondary infection, which may be very difficult to control if the accompanying allergy is not recognized.

THERAPY

The discussion of the therapy of sinus disease in children can well begin with a statement of two things not to do. In the first place, the temptation to resort to tonsillectomy should be resisted until it is clear that the operation is called for because the tonsils are diseased, not merely enlarged; as an English writer said caustically, enlarged tonsils should be removed from the medical terminology, not from the pharynx. Piness and Miller[4] are also very emphatic on this point, stating that the extent to which tonsillectomy is recommended in allergic sinusitis is a measure either of the degree to which the allergic background is unrecognized, or, if it is recognized, the de-

gree to which physicians are ridden by the tonsillectomy complex. The blame for the mistaken therapy they found was rather evenly distributed. In 84 cases they studied from this standpoint, they found that the operation had been advised by general practitioners in 22 per cent, by pediatricians in 32 per cent, and by otorhinolaryngologists in the remainder; evidently we are all to blame.

Griffiths[8] prefers to leave the tonsils *in situ* until after the sinuses are cleared of infection, because experimental evidence suggests to him that they function as filters for toxic substances originating in the sinuses. Mitchell[9] gives the same advice. Whether or not they are right, an examination to determine that the sinuses are free from infection may prevent some tonsillectomies and may forestall disappointing results if the operation is done.

The second prohibition in the treatment of sinuses is against the modern temptation to result to chemotherapy as a routine. The temptation should be resisted until it is clear that the case is one which would be benefited by this measure. Allergic sinusitis is not in this category. In fact, sinus disease is not a specific disease, as Fabricant[10] has pointed out, and success is not to be expected when it is treated as if it were. In his recent comprehensive study of the subject he emphasizes that alkaline preparations of sodium sulfathiazole solution injure the nasal mucosa, destroy the cilia and impair their function, deviate from the physiologic nasal pH, and disorganize the olfactory epithelium, quite aside from their possible toxic effects and the possible induction of a sulfa-resistant state, for which later the patient may have small reason to be grateful. If chemotherapy is the treatment of choice—the merits of penicillin in sinus disease are still to be tested—the choice of drug, mode of administration and dosage must be carefully related to the type of infection present and to the patient's age. It should also be remembered that sulfa drugs are bacteriostatic, not bacteriocidal, and that they do not take the place of drainage and of other indicated surgical procedures.

Sinus disease of allergic origin must be treated first of all from the standpoint of the allergy; even if secondary infection is superimposed, its correction will not be permanent unless the underlying allergy is corrected. Obstructing polyps, which are usually regarded as a manifestation of allergy, should be removed by an atraumatic technic.

In both the allergic and pyogenic varieties of sinusitis the basis of treatment is a hygienic rule of life, with respect to environment (which should not be too hot or too dry); adequate clothing; a balanced diet, without an excess of carbohydrates and with adequate vitamins, especially vitamin A; regular hours of sleep and rest; and correction of anemia, hypothyroidism and similar conditions. If upper respiratory infections develop, particular care must be used to prevent further aggravation of the sinus disease. A change of climate is usually impractical and is necessary only in the exceptional case.

Aside from these general measures, the principle of management should be to treat the patient as an individual, and to make local measures as simple as possible. Local applications of various sorts, irrigations, vaccine therapy (the results of which are most uneven), short wave diathermy, infra-red therapy and hot-air treatment may be used on the same indications and with the same precautions as in the adult. If these measures, after a reasonable trial, prove unsuccessful, drainage should be carried out, by the simplest possible method. Radical operation is required only in extreme cases. The Caldwell Luc operation is advocated by some authors, on the ground that the intranasal approach is unsurgical, but others believe that the risk of injury to unerupted teeth is too great if any other method will suffice. Exenteration is justified in sphenoid or ethmoid infections only if the sight is threatened and when all other causative factors have been elimi-nated. Suppurative complications require

surgical treatment according to the usual indications.

Conservative measures are reported to be successful in from 80 to 98 per cent of all cases. On the basis of even the lower figure they should be given a fair trial before any other therapy is considered, including tonsillectomy.

SUMMARY

1. Two facts are generally overlooked in respect to sinus disease in children, namely, that it is very frequent, and that it is very frequently of allergic origin.

2. According to its origin, sinus disease in children is classified pathologically as purulent and allergic, or as intumescent, catarrhal, and purulent. The manifestations depend upon the origin and pathology.

3. Although diagnostic procedures are sometimes difficult, because of the age of the patients, sinus disease in childhood should be investigated by the same procedures as are employed in adults. Certain diagnostic pitfalls are emphasized.

4. Therapy should be as conservative as possible, with tonsillectomy carried out only when the indications for the operation are clearcut. Chemotherapy is useful only in selected cases and does not take the place of drainage.

5. Sinus disease of allergic origin must be treated first of all from the standpoint of the allergy.

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THE NORMAL AND ABNORMAL PHYSIOLOGIC EFFECTS OF TROPICAL CLIMATE*

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It is well to remember that many people who enter the tropics escape tropical diseases if they are at all careful, but no one can escape the climate. In fact, there is no doubt that thermal regulation is the major problem confronting man when in tropical climates. He immediately has difficulty in eliminating excess body heat efficiently due to the high humidity and high temperature of the environment. The higher the humidity and the closer the environmental temperature to body temperature, the greater this problem becomes. Obviously, when the rate of heat production is increased by exertion, the need for elimination of heat increases. As will be seen below, the influences of tropical environment on body heat are responsible for many physiologic and morphologic changes. Some of these are responsible for a sense of ill health and impairment of efficiency; a complaint of almost all inhabitants of temperate or cold climates who visit the tropics, as well as of many natives of the tropics. This sense of ill health and decreased efficiency is particularly marked when prolonged strenuous physical effort is maintained in the hot and humid climates—conditions under which heat production is great and the rate of elimination not great enough to maintain the normal level of body temperature.

BALANCE OF BODY TEMPERATURE

The average human body temperature is 98.6° F. In order to maintain this temperature, an equilibrium is established between heat production and heat elimination when the body temperature reaches 98.6° F. If the subject finds himself in a cold environment so that the loss of heat becomes greater than that of heat produc-

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tion, the body temperature tends to fall. In order to compensate for this tendency the rate of heat production may increase and mechanisms immediately come into play to reduce the rate of heat loss. Should a subject enter a tropical or subtropical environment, the efficiency of heat elimination is reduced and body temperature tends to rise. In order to maintain normal body temperature the rate of heat production is reduced and mechanisms develop which increase the rate of heat elimination. Some of the mechanisms involved, especially in tropical and subtropical environments, will be discussed very briefly:

HEAT PRODUCTION

Heat is liberated in the process of metabolism of foodstuffs. All living cells liberate heat during the course of activities associated with the state of life. Certain cells are greater producers of heat than others, namely, muscular cells and glandular cells, particularly the liver. During periods of great muscular activity heat production may exceed elimination and the body temperature may reach 104° F. or more. In the digestion and assimilation of food, especially proteins because of their specific dynamic action, heat is liberated in large quantities. Again, everyone is well acqainted with the increased rate of heat production with shivering and chattering of the teeth upon entering a cold environment.

The rate of heat production under basal metabolic conditions varies especially with the size, age and sex of the subject. Detailed values are readily available in tables of metabolism. For example, at the ages of 25 to 29 years, males liberate 40.3 calories per square meter per hour and females 36.6. As mentioned previously, with increased cellular activity, as with exercise, these values are increased manifold.

HEAT LOSS

Heat is lost from the body through: (1) Radiation, convection and conduction; (2) evaporation of water from the surfaces of the skin, and respiratory tract; (3) raising of inspired cold air to body temperature; (4) excretion of CO_2 by the lungs, and (5) warm urine, feces and other excreta and secretions.

Most of the heat (about 90 per cent) is lost through vaporization of water, radiation, convection and conduction.

CONDUCTION

Heat lost by conduction depends upon the difference between the temperature of the surface of the body and that of the environment. The greater the difference the greater the rate of exchange. If the body temperature is higher than that of the environment heat will be lost from the body. The greater this difference the greater the loss. If the temperature of the environment is greater than that of the body, heat will be transferred from the environment to the body by conduction. The nature of the medium or environment in which the body is found will also influence the rate of heat transfer by conduction. Air is a poor conductor of heat while water is a good conductor. The latter is about 30 times a better conductor than the former; for example, a subject in a bath of cold water will lose heat by conduction thirty times more rapidly than in air at the same temperature. The denser the air or the greater its water content the greater its conductivity. Dry warm air therefore is not as good a conductor of heat as cold moist air. In the usual subtropical and tropical environments the temperature of the environment approaches that of the body so that very little body heat can be lost by conduction. In some desert temperatures heat is transferred from the environment to the body.

RADIATION

Heat is transferred by radiation from the hotter body to the colder one. If objects, such as furniture, walls, ceiling and the like in an environment are cooler than the surface of the human body, heat will be lost to those cool bodies by radiation. If the surrounding objects are warmer than the surface of the human body, heat will be transferred from them to the body of man. Everyone is acquainted with transfer of heat by radiation from a red hot stove to his body. Hardy has shown that the human skin, regardless of color, radi-

ates like a black-body*. This is little influenced by the presence of water vapor and CO_2 in the layers of air next to the skin. A person will feel comfortable in a room with cold walls even though the temperature of the air is warmer than the body temperature, for more heat will radiate from the body to the walls than will be conducted from the warmer air to the body.

When bodies at different temperatures are near each other heat will be transferred from each to the other by radiation until they all reach the same temperature or a state of thermal equilibrium. The rate of transfer by radiation will be determined by the size of the bodies, their respective temperatures, the emissivity of the bodies, and the absorption, reflection and transmission qualities. These all important factors can be found discussed in detail in textbooks on physics. All human skins are fairly good conductors of heat and excellent radiators. The emission, infra-red transmission and reflection spectra of human skin are essentially that of a black-body radiator (Hardy).

In subtropical and tropical climates with the daily hot sun and the surrounding hot roads, streets, and buildings, it is difficult for a person to find a place in which he is not absorbing heat by radiation, at times in large quantities. This is especially true when on a dry, hot sandy desert.

CONVECTION

The transfer of heat to or from a fluid (liquid or gas) flowing over a surface of a hotter or colder body is by the process known as convection (Brown and Marco). Convection is termed free or natural when circulation is produced by differences in density produced by differences in temperature within the fluid, and forced convection when circulation is produced by mechanical means, such as a fan or pump.

The more rapid the flow of the fluid (usually air in the case of the human body) and the greater the quantity the more the rate of heat transfer. Furthermore, the greater the capacity and conductivity the fluid medium for heat the greater the transfer. For example, more heat can be transferred by flowing water than flowing air. In man, convection of heat by air is involved more often than by water. Flowing air that is warmer than the human body will transpose heat to the body, while cooler air will remove it.

In subtropical and tropical climates flowing warm air is not as effective in cooling man, since the air temperature is usually near that of the body. In hot desert air it may even transfer heat to the body. These effects are even greater when water is concerned. It is well to remember that evaporation is not considered in the above discussion of air currents.

EVAPORATION

The loss of body heat by the vaporization of water from the lungs and skin is important. This method of heat elimination becomes extremely important when heat production is rapid or the environment is such as to reduce heat loss by the methods described above. For each gram of water vaporized 0.58 calorie of heat is removed from the body. Perspiration is classified into two types:

a. *Insensible perspiration*: This includes all water lost from the body insensibly, that is, water that evaporates as soon as it reaches the surface of the body. This includes water that diffuses through the skin, water that is secreted by the sweat glands at a rate slow enough to vaporize before a visible quantity can accumulate, and water that vaporizes from the lungs.

b. *Sensible perspiration*: This includes visible sweating.

Insensible perspiration: The rate of insensible perspiration correlates very highly with the basal metabolic rate. It varies with the size of the subject, but averages about 28 grams per hour. The respiratory portion is about 40 per cent of the total, although the values found in the literature vary considerably. Obviously, the amount of water lost, insensible from the skin or

*A true black-body is one that emits the maximum radiation at a given temperature. The adjective "black" has nothing to do with the color of the body (Brown and Marco, Introduction to Heat Transfer, McGraw-Hill Book Co., Inc., N. Y. and London, 1942).

lungs, increases with the environmental temperature and decreases with increases in the humidity. Dehydration tends to decrease insensible perspiration and hydration tends to increase it. Hot and dry air currents will increase insensible water loss from the skin. By definition, profuse sweating may be included (vide supra) as insensible perspiration when the rate of evaporation is such as to ensure a dry skin. For this reason it seems to be preferable to consider only water lost from the body by diffusion as insensible perspiration. A subject with ectodermal dysplasia without sweat glands may lose 400 calories in 24 hours by insensible perspiration which is by diffusion only of water through the skin.

The heat eliminated by insensible perspiration varies considerably, ranging from 19 to 32 per cent of the total heat loss under basal conditions. The mean is about 25 per cent of the total body heat loss under basal comfortable environmental conditions. Heat lost by insensible perspiration does not vary greatly even under subtropical and tropical conditions and is of little importance when heat production is rapid, such as during strenuous exercise.

Sensible perspiration: Sensible perspiration is emergency sweating. It comes into action whenever heat production is very rapid and heat elimination by radiation, conduction, convection and insensible perspiration are inadequate to cope with heat production or when the environment is hot so that the temperature of the body is near or lower than that of the surrounding atmosphere. Under such circumstances sweating takes place. If the humidity of the atmosphere is low and especially if there are air currents, the sweat water is vaporized, cooling the surface of the body. If the humidity of the air is high, evaporation is impaired and the sweat rolls ineffectively from the surface of the body. Sweating may reach one liter or more per hour under hot environmental conditions during exercise. The evaporative powers of the atmosphere determine the effectiveness of such rates of sweating. In a dry windy desert atmosphere all of the sweat water may be vaporized as rapidly as it exudes from the sweat pores. Obviously rates of sweating as great at one liter per hour can easily upset water and electrolyte balance and often does so to such an extent to become a serious medical problem under certain circumstances of warfare and industry. Dill and his associates have shown that persons residing in a hot and humid environment may double their sweating ability after some time, a phenomenon of adaptation, training, or acclimatization.

A man can tolerate atmospheric temperatures of 240 to 260° F. if it is dry and sweat can evaporate freely. Such a temperature may grill a beefsteak (Best and Taylor), while man cannot tolerate a temperature of 120° F. if it is so humid that sweat only drips off the surface of his skin. With profuse sweating and strenuous exercise in a dry environment most of the body heat loss will be through the vaporization of sweat.

Sweat is the most dilute fluid secreted in the human body. Its specific gravity varies between 1.002 and 1.003. It may become concentrated, for as the water evaporates the solutes accumulate. Sweat is essentially an aqueous solution of sodium chloride although it contains urea, uric acid, some vitamins, various non-protein nitrogen substances and a very small amount of protein. Eccrine glands secrete little if any protein in the sweat, while the apocrine glands found mainly in the axillae and perianal regions of man, secrete sweat containing larger amounts of proteins. The horse secretes sweat rich in protein, particularly albumen. This is evidenced grossly by the foaming of his sweat where it is stirred in his hair by the harness, saddle or intertrigenous regions. A horse may develop hypoproteinemia from profuse and prolonged sweating. In man profuse sweating often results in hypochloremia. He may lose 0.5 to 1.0 gram in his sweat per hour with profuse sweating. Sweat is usually acid, the pH varying between 4.8 and 8.4 in a manner very similar to urine.

Claims have been made that there are racial differences in sweating. For ex-

ample, it is claimed by some that in the negro the sweat smears over the skin more thoroughly thus making evaporation more effective and thereby aiding in the ability of the negro to cope with tropical climates. Such claims have not stood the tests of careful observations. In fact no definite differences that may explain racial peculiarities in acclimatization to tropical climates can be ascribed to sweating.

Sweat glands: Glands of the skin are composed of two types, *sudoriferous* and *sebaceous*. The former secrete sweat and are extremely important in body temperature regulation. The number of glands vary from area to area over the body, being entirely absent from the concave surface of the concha of the ear, labia minora, inferior surface of the labia majora, glans penis and the inner surface of the prepuce. The regional variations per square contimeter are: 400 glands on the flexor surfaces of the hands and feet, 200 on the forehead, 180 on the chest and arms and 90 on the legs and much less in number on the eyelids and convex surface of the concha. The sweat glands are of two types, eccrine which secrete clear sweat and apocrine which are limited to the areola, genital and circumanal regions.

Claims have been made that natives of the tropics and especially negroes have a greater abundance of sweat glands making them more capable to cope with hot and humid environments. This claim likewise has not been fully substantiated.

RAISING OF INSPIRED AIR TO BODY TEMPERATURE

The amount of heat lost by the process of heating of inspired air depends upon the temperature of the air inspired. The colder the air inspired the more will be the heat loss. The heat loss in subtropical and tropical environments by this means is not great. In fact, heat may be accumulated in hot desert temperatures during the day. Under resting conditions in a comfortable environment this amounts to 2 to 3 per cent of the total heat loss. With rapid respiration and low air temperatures this may be increased slightly. Air has a low capacity for heat and therefore is a poor medium for the transfer of heat.

6. *Loss of CO_2 from the lungs* accounts for only 3 per cent of the total heat loss.

7. One to 2 per cent is lost through the passage of warm *urine and feces*.

PHYSIOLOGIC MECHANISM CONCERNED WITH THERMAL REGULATION

A *thermal center* is located in the diencephalon or, more precisely, the hypothalamus. This center is concerned with the setting of the temperature level of the body. It is the main center through which the mechanisms of heat production and heat elimination are integrated so as to maintain a given body temperature, 98.6° F. in the average normal subject. There are thermal *spinal centers*. The nature of the function of these centers is unknown. Injury to the hypothalamus will result in fever. Severance of the nerve pathways to the cerebral and spinal centers results in disturbances in body temperature. These centers act through the mechanism of the vasomotor mechanisms, sweating, metabolism, and others. In infections, mainly through the thermal centers, the mechanisms are integrated in such a manner so as to produce fever. Antipyretic drugs likewise owe some of their effects through actions in these centers. It is beyond the scope of this presentation to enter into any detailed discussions of these mechanisms.

When a person enters a subtropical or tropical environment his body temperature tends to rise. In order to overcome a decided rise, physiologic mechanisms are set in motion in order to increase the rate of heat elimination by radiation, convection, conduction and evaporation. There is a peripheral vasodilation of the skin vessels increasing the quantity of blood reaching the surface of the body to liberate heat by convection, conduction and radiation. There results a redistribution of the blood so as to decrease the visceral volume and to increase the volume in the skin. The spleen, for example, constricts. The heart rate increases and the cardiac output per minute increases, thereby recirculating the blood rapidly and frequently through the interior

of the body and through the muscles where the blood collects the heat liberated by metabolic processes and then to the surface of the body to discharge its heat load. Furthermore, if the individual has good judgment he will place his body in a cool draft of air to profit by the maximum benefits of the factor of convection. He will also avoid sources of heat such as engines, fires, and the like. Sweating begins if his skin and body temperatures continue to rise. This increases the rate of heat loss by evaporation. Should these mechanisms fail to keep the body temperature at the normal level then heat production must be reduced by the reduction of muscular exertion. Apparently after several months' sojourn in the tropics the basal metabolic rate decreases, a physiologic adjustment to reduce the rate of heat production.

During acute elevations in environmental temperature and humidity the changes described above are exaggerated. The respiratory rate and volume of tidal air are increased which increase the rate of heat elimination through the respiratory tract. This is not so great among people living in the tropics. When the mechanisms concerned with heat elimination fail, then heat exhaustion and heat stroke result. These syndromes are discussed below.

There are special apparatuses that have been developed to express quantitatively the influence of the environment on heat elimination. Among the many such instruments is the kathathermometer of Leonard Hill and the cooling ball of the Germans. These methods integrate heat loss by radiation, conduction, convection and evaporation under given environmental conditions. The usual dry bulb temperatures usually reported in the daily newspapers fail greatly in expressing the comfort of the weather for that day. Such inadequacies are fulfilled only in part by the above special instruments. These instruments do not take into consideration the factors of exercise, previous acclimatization, training, or other factors related to the so-called personal equation.

PHYSIOLOGIC EFFECTS OF TROPICAL CLIMATES

It is well known that a person in a hot and humid environment is not comfortable and satisfied. This discomfort probably is a result of physiologic compensatory mechanisms, such as cutaneous vasodilating, sweating, and tachycardia, integrated to maintain a normal body temperature. The mechanisms concerned with the peculiar feeling of discomfort are difficult to explain. Even though his body temperature is no different from that in a cool environment the mere fact that his physiologic heat eliminating mechanisms are active makes him uncomfortable. It was found that this feeling is markedly lessened if an arm or a portion of the body is cooled in water to facilitate the elimination of body heat. These sensations of discomfort are probably the result of many factors, chemical, physical and psychic. This is more pronounced when there are acute and marked environmental changes than when the changes are gradual and remain for a prolonged period such as are found in entering and remaining in the hot and humid tropics. Although the mechanisms involved are not well understood certain changes concerned in a large part with acclimatization occur in tropical climates. The mechanistic nature of the changes are little understood. Some of them are:

Growth: There are no detailed groups of studies on the influence of tropical climates on growth. The data available at present indicate that growth in general is impaired by tropical climates. The body tends to become linear, the weight decreases and the ratio of surface area to body volume increases. This facilitates cooling of the body. Natives of the tropics tend to be small. Evidence suggests a decrease in the reproductivity of man living in the tropics. Sexual maturity is reached earlier in the tropics than in the colder climates. Sterility among women is greater with a predisposition to menstrual dysfunctions and abortions. Laboratory studies with experimental animals grown under tropical environmental conditions support the above conclusions. It is, however, difficult to separate distrubances that arise from climatic conditions *per se* and those due to

chronic infections, malnutrition and psychic maladjustments so common among natives and emigrants in the tropics. The growth of the hair as well as the skeleton is impaired. The rate and quantity of growth of hair is impaired by tropical climates. This is particularly true among fur bearing animals. This would be a necessary adjustment to facilitate cooling of the body.

There is an increase in the *pigmentation* of skin and the body in general in the tropics. The stimulus for this is derived in a large part from the ultra-violet light and heat from the sun present in great quantities at all times of the year. The pigment protects the underlying structures from injury by the sun's rays. The endocrine functions are disturbed by a tropical climate. As mentioned above, menstruation appears early and may be disturbed. In rats the thyroid gland has been shown to decrease in weight. The adrenals as well as all other organs decrease in weight at least proportionally with the decrease in body weight under tropical climatic conditions. Almost all reports on studies of the basal rate of metabolism indicate a relatively low rate both in natives and Europeans who remain in the tropics for several months. The mechanisms concerned with the metabolic change remain unknown.

The *nervous system* is particularly affected by the tropical environment. It is unknown as to how much of this is related to temperature and humidity *per se*. It is well known that most colonists and colonial workers assigned to tropical regions for long periods of time are frequently unhappy. It is unknown how much of this is due to the sudden change in things socially, to the ever present and annoying insects, the heat and humidity, the uneducated natives, the lack of stimulating influences of competition in business and society, homesickness for friends and relatives many miles away, and many other such factors. In the tropical regions of Australia where European emigrants have lived for generations "tropical neuroses, irritability or neurasthenia" are not nearly so common and pronounced. It is a well known physiologic fact that a steady stereotype stimulus fails to influence the animal after some time. One becomes so accustomed to an odor that he soon fails to be aware of its presence, or he becomes accustomed to a steady hum of a motor or the tick of a clock and is soon unaware of their presence. The same is true for the heat and humidity and lack of seasons of the tropics. In the temperate zones, changes in the seasons and sudden changes in weather are stimulating.

Tropical climates are free from very much weather and therefore are, at least, not stimulating and may even be depressing. As pointed out previously, when a person is in a hot and humid environment and must make the necessary physiologic adjustments, particularly sweating to assist in the dissipation of excessive heat, he is uncomfortable, unhappy and mentally ill at ease even though his body temperature is maintained at normal levels. This probably contributes to the mental as well as to the impairments in physical efficiency. It is unknown what part is due to central nervous system fatigue and fatigue in the autonomic nervous system especially concerned with the adjustments necessary for thermal regulation. Much has been written about such matters but little has been settled. Mental efficiency is impaired by the tropics although the causes are unknown.

There is a definite disturbance in *water balance* in the tropics because of the extreme importance and high development of the sweating mechanisms. As stated above, sweating is of utmost importance in thermal regulation in tropical climates. Without sweating fairly healthy life is impossible in these hot environments. A man may lose as much as one liter of sweat an hour. This results in a marked disturbance in water and electrolyte balance if the water and salts are not replaced. Much salt, mainly sodium chloride, is lost in the sweat and urine with marked sweating. If the water is not replaced the urine volume decreases and becomes highly concentrated. In the management of such people it must be re-

membered the daily water and salt intake and output must be equal if an equilibrium is to be maintained. Such an equilibrium is necessary for good health (vide infra). There is no definite evidence to support the idea that the blood is "thinner", that is, of lower specific gravity, among residents of the tropics. In fact some observers believe the converse is true, especially during very hot periods when sweating is marked. It is well to remember that the excretion of highly concentrated urine may result in variable degrees of kidney damage with the appearance of abnormal amounts of casts, erythrocytes, and albumin. Most physiologists agree that the formation of much weak (low specific gravity) urine will not injure the kidneys but the excretion of concentrated urine for prolonged periods will. The climate of the tropics predisposes to the latter unless man is attentive to his water requirements. Nephritis in general appears to be less prevalent and fulminating in the tropics. This may not be true since medical records and methods in the tropics are not as highly developed as in the cooler climates. The blood NPN and chloride have been found to be elevated in many normal people residing in the tropics. These changes are said to be due to a lack of sufficient water for urinary excretion. In the very warm and trying periods, the non-protein nitrogen was found to be elevated among people of the tropical parts of Australia but was normal at other periods or among Australians living in the cooler parts of the continent. The various components of the non-protein nitrogen fraction were proportionately increased.

The *acid-base balance* tends to be shifted to the alkaline state contrary to most expectations. The alkaline reserve is unchanged or increased. Acid-base studies, however, are still wanting. The blood sugar level tends to remain at lower levels than those of people living in cooler climates. The blood phosphorus levels likewise tend to be slightly lower than those of inhabitants of the temperate zones. The lipoid constituents of the blood are also quantitatively different among inhabitants of the tropics. Antagonistic levels for lecithin and cholesterol in the blood were found. High levels of lecithin were associated with low levels of cholesterol and vice versa. The cholesterol levels obtained in Queensland were normal or elevated during the very hot seasons. The influence of nutrition upon some of the results reported remain to be evaluated.

The *formed elements of the blood* are not influenced by tropical climates. The old idea that everyone in the tropics suffered from an anemia which contributed to his sallow complexion has been shown by careful and properly controlled studies to be erroneous. The erythrocyte and hemoglobin contents of the blood are no different from those of inhabitants of cool climates. The causes for the sallow skin in the presence of a normal erythrocyte count and normal hemoglobin level have been advanced but not proved. The sedimentation rate of the erythrocytes is increased by the tropics. Numerous time consuming studies of the white blood cells have been reported. Most reports indicate a decrease or a normal number. Some investigators have shown the subnormal counts to return to normal levels when the persons concerned returned to cooler climates. Many have studied the polymorphonuclear leukocytes as classified by the Arneth index, reporting a shift to the left. A study of these reports leaves the impression of a gross failure of the observers properly to control such factors as nutrition and low grade chronic infections, especially of the gums and gastrointestinal tract.

The *blood pressure*, systolic and diastolic, is little influenced by the tropics. Strenuous exercise produces a marked and rapid rise in blood pressure followed by a drop below the previous basal level.

The pulse rate remains about that for cool climates after acclimatization but tends to be high upon first entering the tropics. The blood volume tends to increase slightly in a hot and humid climate. There is a need for a more careful study of the body fluids.

Respiratory rate and volume apparently are little influenced by a tropical climate al-

though the reports have varied considerably, some indicating a decrease and others an increase. *Gastrointestinal activity* is not materially different among natives of a tropical climate and those of a temperate climate.

The above physiologic discussions indicate that the physiologic state of natives of a tropical climate is not very different from that of persons in cool climates. Certain changes concerned with heat transfer are certainly definitely present, among which are a decrease in body weight and an increase in surface area of the body, increase in pigmentation, increase in sweating and the ability to sweat, and a decrease in the rate of metabolism and unnecessary exertion. Most of the studies from the tropics appear to fail to control such factors as malnutrition and low grade chronic infection related not necessarily to the tropics *per se* but to education, public health and economics. Furthermore, there is a great tendency among observers to fail to differentiate physiologic phenomena associated with acute elevations in environmental temperature and humidity from those in natives of the tropics or emigrants who have been in the tropics for several years. Such differentiations are necessary and should be considered very carefully before any sort of generalizations are made concerning the influence of a tropical climate on man.

CLINICAL STATES DUE TO TROPICAL CLIMATE

The hot and humid environment encountered in the tropics may produce three clinical states concerned with disturbances in the heat-regulating mechanisms or electrolyte balance or both. These are: heat cramps, heat exhaustion, and heat stroke.

Each syndrome is discussed separately except for the differential diagnosis and treatment. These latter two phases of the syndromes are presented jointly since there is so much in common.

HEAT CRAMPS

Heat cramps are associated with painful spasms of the voluntary muscles following muscular activity in a hot environment. There are many terms that have been applied to this process such as stoker's cramps, mill cramps, cane cutter's cramps, miner's cramps and fireman's cramps. Heat cramps are by far the most common of the three syndromes associated with a hot environment.

The *incidence* of heat cramps varies considerably with the occupation. Natives of the tropics have learned to reduce their muscular activity during the hot periods of the day. This decreases the amount of heat produced and thereby reduces the need for the elimination of extra heat. Nevertheless, under certain circumstances such as in mines, marching of soldiers, and in certain industries, heat production continues at a high rate in an environment in which the elimination of heat is impaired. In the British and American Army the number of soldiers suffering with heat cramps increases during marked activity of the men. Heat cramps are common in certain industries even in temperate climates.

The *mortality rate* is unknown in heat cramps. The disease may be fatal, but is essentially benign if the proper therapeutic measures as instituted promptly. Most fatalities are the result of some concomitant disease or complications resulting from neglect.

The *climate* is a significant factor in the production of heat cramps, but in the case of the tropics as well as temperate zones the immediate environment may be even more important. Environmental temperatures that reach or exceed the level of body temperature make it difficult to eliminate an excess of body heat. In fact, heat is transferred to the body from the environment by conduction, radiation and convection. Heat can be eliminated only by the evaporation of sweat. If the subject is exercising considerably, the load placed upon the vaporization of sweat is increased further. This is seen in firemen working in firerooms at temperature of 112°F. or men working in sugar refineries at temperatures between 112° and 135°F.

When the relative humidity is high the evaporation of sweat is impaired, thus reducing the efficiency of heat loss by sweating. Working under such conditions re-

sults in profuse sweating with marked loss in water and electrolytes.

Organic disease, alcoholism, poor hygiene, fatigue, malnutrition and generally poor health predispose to heat cramps. This should be remembered when people are to enter environments and circumstances in order to reduce the tendency to heat cramps. Repeated and recent attacks of heat cramps predispose to other and more severe episodes. Lack of acclimatization, likewise, predisposes to heat cramps.

The *pathology* associated with heat cramps has not been carefully studied because so few necropsies have been performed. Vascular congestion of the lungs and gastric mucosa has been described.

The *pathogenesis* of heat cramps is better understood today. The loss of sodium and chloride ions from the body fluids through the sweat glands and kidneys is the principal factor responsible for the production of the heat cramps. The blood and tissue fluids have about an equal reduction in these electrolytes. When critically low levels are reached cramps result. Persons who are sweating profusely over prolonged periods of time are losing water and salt in such a manner as not to reduce the concentration of the electrolytes. Drinking a considerable amount of water without the ingestion of salt reduces the concentration of the sodium and chloride ions thus precipitating heat cramps.

Disturbances in carbohydrate metabolism may be related to the production of heat cramps, but direct evidence to support any such relationship is wanting.

Incidentally typical heat cramps have not been produced experimentally in men or animals.

The *clinical picture* offers as a rule little diagnostic difficulties. After exposure to a hot environment while at work the victim begins to experience gradual involuntary spasmodic contraction of his skeletal muscles. The flexor muscles of the fingers are usually affected first. The cramp occurs every few minutes. Sometimes the larger muscles as those of the legs and arms and abdominal wall are affected instead of the smaller ones. When the abdominal muscles are involved the picture of an acute abdomen may be simulated. The muscles remain contracted for a variable period of time, usually not over three minutes. The contractions are associated with severe pain which may be almost unbearable at times. The contractions and pain varying a great deal in degree, develop slowly to reach a peak and then subside with relief of pain. Sudden movement, jarring and breezes may precipitate a spasm. The pain may be so severe that patients prefer death to another episode—still at times the contractions and pain are so mild that the subject continues to work with them without even seeking medical attention. As a rule there are no other pertinent manifestations. The pupils dilate with each episode of muscular spasm. The patient may be sweating and the skin is cold and clammy. The pulse may be slightly accelerated, but is usually normal. There may be headache, nausea and vomiting are rare.

On *examination* the affected muscles show fibrillary twitchings which make the underlying tissues appear to be alive with a mass of snakes. Palpation, firm or mild, of the muscles may precipitate violent contractions and severe pain. During the contractions the muscles characteristically become stony hard. These contracted muscles do not respond favorably to massage.

Except for the muscle findings, the remainder of the examination is essentially irrelevant. The temperature is normal or only slightly elevated. The respiratory and pulse rates may be slightly increased. When the abdominal muscles are involved the picture may resemble an acute abdominal syndrome such as acute peritonitis, appendicitis, and the like. Between the periods of muscular spasm this picture disappears. The Chvostek and Trousseau signs are characteristically absent. The neurologic examination is normal. Any mental disturbances are the result of the pain.

The cramps may be ushered in by mild vertigo, headache and feeble muscle twitching. They usually follow one or more hours in a hot environment in a susceptible per-

son and several days in others. At times the cramps appear several hours after leaving work. The mechanisms for the pains are unknown but are probably the result of local effects within the muscles of dsiturbances in electrolyte balance. The pain appears to originate peripherally in the muscles and not in the nervous system. Cramps are known to have included almost all voluntary muscles. Spasm of the heart occurs rarely if at all. Flexion, pressure, knocks, cold air or water and almost any form of direct stimulation of the muscles may illicit a spasm of the muscles in severe cases. Profuse sweating precedes these attacks and may continue paroxysmally during the periods of actual pain and muscular spasm.

The *laboratory examinations* usually show evidences of dehydration and sodium chloride loss. The blood proteins are elevated, the serum proteins may reach 12.7 grams per cent, resulting mainly because of an elevation in the globulin fraction. In fact the albumin fraction may be depressed. The elevations are due in part to anhydremia. With improvement and hydration the levels return to normal. The hemoglobin and erythrocyte concentrations increase with the dehydration to levels comparable to those of the blood proteins. The erythrocyte count may reach 6,500,000. Total blood volume is probably decreased slightly with dehydration, but adequate studies are wanting.

The acid-balance is changed somewhat. All ions are changed consistently except the bicarbonate ion. The latter may be increased, decreased or normal. It may drop to below 22 m. eq. The pH varies considerably, from 7.08 to 7.48. It is usually below 7.40. The acidosis is most probably due to a depletion of base rather than an accumulation of organic acids. There is an increase in inorganic phosphate. The cause is unknown, but the increase may be 1 to 3 m. eq. It is not dependent on oliguria. Lactic acid is little changed from the normal.

The changes in sodium and chloride are consistent and significant and have been stressed in mechanistic explanations of heat cramps. The blood levels are lowered. The chloride levels may read 54 m. eq. (normal 99 to 108 m. eq. or 550 to 600 mq. per 100 c.c.) and are usually below 100 m. eq. The sodium level may read 121 m. eq. (normal 153 m. eq. or 335 mg.) and usually is below 140 m. eq. It is the only inorganic base that is lowered in heat cramps. The potassium levels may be increased 1 to 3 m. eq. These changes result in an alteration of sodium and potassium ratio, a possible contributing factor concerned with the muscle spasms. The calcium level of the blood is not decreased but may be increased. A maximum concentration of 14.3 m. eq. or 14.3 mg. per 100 c. c. has been reported. A part of this change may be explained by the change in blood proteins. The serum magnesium is not changed. The blood sugar is not changed.

The non-protein nitrogen content of the serum is usually elevated 10 to 25 mg. per 100 c. c., levels of 108 mg. being reached at times. A urea nitrogen level of 120 mg. has been reported.

The white blood cell count is usually increased 4,000 to 6,000. A value of 18,000 has been reported. This like the change is non-protein nitrogen is a rather constant finding in heat cramps.

In summary, the changes in the blood acid-base balance are quite large. The increase in serum protein and cell volume are of the sort seen with severe dehydration. The degree of serum sodium, potassium and phosphate changes are comparable only to that of adrenal deficiency. The rise in blood calcium is comparable only to that encountered in hyperparathyroidism.

The urine is reduced slightly in quantity 400-800 c. c. per day, but there is no marked oliguria. The pH of the urine is usually about 5.0. The concentration of chloride is below 40 m. eq. per liter, although occasionally it is greater than this. With therapy and the administration of water and salt there is a relative absence of sodium and chloride in the urine. During such a period 40 or more grams of sodium chloride may be retained in a three

day period. The potassium excretion in the urine is also decreased. An increased urinary excretion of phosphate and creatine suggests increased phospho-creatine breakdown in this disease. The total nitrogen excretion may be increased. Albumin may be found in abnormal amounts in the urine.

HEAT EXHAUSTION

Heat exhaustion or heat collapse is characterized by profuse sweating, weakness, vertigo, syncope, pallor, low blood pressure, thready rapid pulse, a subnormal, normal or slightly elevated temperature and the general picture of peripheral circulatory collapse or shock. This is not a serious syndrome as a rule, the mortality rate being low.

The *incidence* of heat exhaustion varies considerably with many factors. It is more common during very hot weather lasting long periods of time. In the tropics and especially tropical desert areas particularly when muscular activity is strenuous for long periods in the hot sun, such as soldiers on the field of battle or maneuvers, heat exhaustion is common. In some of the subtropical regions of the United States, Iraq or Iran the incidence is high during the summer. Poor acclimatization, poor health, restriction of water and salt intake increase the incidence. Colonists from the cooler areas of the earth suffer from heat exhaustion if they enter the tropics without taking the necessary precautions in rest, clothing, and avoidance of too much food. In Army establishments located in subtropical and tropical areas heat exhaustion is the main cause for hospitalization of soldiers. It is a common but not serious syndrome produced by hot environments. This is a common problem in industrial medicine where men work near hot furnaces and boilers.

The *mortality rate* in heat exhaustion is low or negligible. Usually the person who remains in the unfavorable environment and fails to heed his symptoms as a warning to seek relief passes into the state of heat stroke and death. Occasionally some patients die of shock. Senility and debilitating diseases predispose to death.

The *climate* is of great importance. The influence of climate on heat exhaustion is essentially the same as indicated for heat cramps. A person working in an atmosphere which is hot and humid strains his thermal regulating mechanisms, resulting especially in profuse sweating with a loss of sodium chloride and water. Working in mines, blast furnaces, boiler rooms and the like predisposes to heat exhaustion. In the tropics an individual not only has difficulty eliminating heat during his working hours but continues with this difficulty throughout the entire day. This leads to final exhaustion and the clinical syndrome described below.

The *pathology* of heat exhaustion is inadequately known since so few people die of this condition alone. Uusually heat exhaustion passes into heat stroke before death. The pathologic findings are not specific and are similar to those described for shock. There may be vascular congestion in viscera with petechial hemorrhages and signs of early degeneration of the parenchymal cells of the viscera.

The *pathogenesis* of heat exhaustion is little known. Like heat cramps, prolonged physical exertion in a hot environment is responsible for the syndrome. A hot climate such as a tropical climate, particularly a desert one, results in heat exhaustion if muscular exertion is even moderate in amount. An inadequate intake of water and sodium chloride seems to be of great importance in the development of this syndrome as in heat cramps. Heat cramps is frequently associated with heat exhaustion. Senility, alcoholism, poor health and debilitating states predispose to heat exhaustion. Unlike heat stroke (vide infra) and like heat cramps there is no collapse of the thermal regulating mechanism. In heat exhaustion the circulation fails, circulatory collapse, after its unsuccessful attempt to provide compensatory physiologic processes to ensure a rate of heat loss equal to heat gain.

The *clinical picture* of heat exhaustion

is not difficult to identify. There are prodromal symptoms which appear in persons working in tropical environments that suggest impending heat exhaustion. These people complain early, one to two days before the onset, of weakness, malaise, profuse perspiration, nervousness, lassitude, giddiness, insomnia, light headedness, vertigo, headache and restlessness. These symptoms increase in severity and in addition there are syncope, nausea, vomiting, cramps, dyspnea, temporary blindness and exhaustion and collapse. The patient may present a shock picture of pale, cool and clammy skin, a rapid thready pulse, and a drop in body temperature below the normal. It is well to remember that the body temperature may be below, at or above the normal level. Should the patient fail to leave the hot environment, stop muscular exertion and enter a cool environment, he may become unconscious and die, or develop the highly fatal state of heat stroke.

The *laboratory studies* show essentially the same findings described above for heat cramps, especially, of course, if heat cramps are well developed with the heat exhaustion. If there is dehydration with hemiconcentration, the hematocrit is elevated and the erythrocyte count is increased. The urea nitrogen levels of the blood may be increased slightly. As a rule the blood chloride level is normal. The urine is normal or may be reduced in quantity and concentrated. As a rule it is normal. At times there may be traces of albumin and abnormal amounts of casts and erythrocytes. When heat stroke is present the laboratory finding of heat stroke will be present (vide infra). In severe cases there is a tendency to acidosis with a definite reduction of the alkaline reserve, evidenced by a reduction in the carbon dioxide combining power.

HEAT STROKE

Heat stroke (sun stroke, heat collapse, thermal fever, heat hyperpyrexia) is a highly fatal syndrome due to a hot environment. It is characterized by extremely high fever and profound coma. It may be preceded by symptoms of heat exhaustion.

The *incidence* varies considerably. It occurs principally in elderly people (60 years or older) and persons suffering with debilitating chronic illnesses. It occurs in environments in which the temperature is high and especially in subjects who are active. Under these conditions they are unable to eliminate sufficiently their excess heat by conduction, radiation, convection and evaporation. This is seen in the mines of the tropics, workers in the hot sun on a humid day without rapid currents of air, or firemen in ships or factories. In the temperate zones many people suffer from heat stroke during severe and prolonged heat waves. There may be periods without any reported cases of heat stroke and others in which the incidence is relatively high. In the temperate zones those who are accustomed to the cool weather as of Boston or Cincinnati are particularly prone to develop heat stroke during heat waves.

The *mortality* rate is very high, varying from 10 to 80 per cent. It is the most serious of the three heat syndromes. The mortality rate is much greater in senile and in chronically diseased subjects than in the younger ones. The earlier and more complete the therapy the better the prognosis.

The *climate*, as in the above two syndromes, is of paramount importance in the production of heat stroke. The rôle of climate in heat stroke is essentially as presented for heat cramps.

It is generally agreed that sunstroke and heat stroke are the same syndromes resulting from disturbances in heat transfer. There is no evidence that sunstroke results from any other factor than that of the radiant heat from the sun, the excessive heat from the sun and environment in general being the source rather than a boiler of a ship, for example. Heat stroke is apt to occur whenever there is a sudden and prolonged elevation in the environmental temperature, and, of course, especially when the individual is exerting himself.

The *pathology* in heat stroke is not well understood. There is marked and early rigor mortis with congestion of the vessels of the viscerae. There may be petechial

hemorrhages in the skin and mucous membranes. The parenchymal cells of many organs may show early signs of degeneration such as cloudy swelling. The blood may be very dark. These patients die so quickly that there is insufficient time for marked degenerative changes to take place.

The *pathogenesis* of heat stroke is little known. Senility and chronic debilitating diseases such as hypertension, arteriosclerosis and diabetes mellitus predispose to the development of the syndrome. When the temperature of the environment exceeds body temperature heat loss by radiation, conduction and convection ceases, and instead heat is absorbed from the atmosphere by these mechanisms. Heat is also being produced by the processes of metabolism. The body heat can only be lost by the evaporation of sweat. Should this loss be insufficient then the body temperature rises to extreme levels. In some reports all patients who have developed heat stroke cease to sweat completely at about the time the syndrome develops. Apparently, the thermal regulating mechanism fails suddenly, resulting in body temperatures of 106° to 107° F. or more associated with the symptoms described below. The reasons for the sudden failure of thermal regulation and the coma are unknown.

It is known that persons who are too warmly clothed and whose clothing fits tightly are prone to develop heat stroke. Such clothing interferes with the loss of body heat.

Disturbances in water and salt metabolism with a resulting dehydration and sodium chloride deficiency may be factors that contribute to the development of the syndrome but they appear to be of minor significance.

The *clinical picture* is characteristic. The prodromal symptoms consist of weakness, vertigo, nausea, syncope, headache, feeling of excessive warmth, mild heat stroke, abdominal pain typical of heat cramps and the symptoms and signs described previously of mild heat exhaustion. Most, and in some series all, patients noted a cessation of sweating shortly before the onset of the heat stroke. All patients had been sweating profusely before the sweating suddenly ceased.

The acute phase of the disease is characterized by a body temperature which is markedly elevated, a dry skin and coma. The body temperature may be less than 106° F., but is usually above that level and may reach 112° F. The patients are usually stuporous or unconscious. There is no sweating, the skin is dry, flushed and hot. There may be a maculopapular eruption over the trunk of the body. The skin may be fiery red and at times marked depression of the nervous system evidenced by flaccid muscles, diminished tendon reflexes and incontinence of feces. The respiration is rapid and deep and stertorous but not labored. Those not in coma show evidence of mental confusion.

Congestive heart failure is not present. There may be coarse rales in the lungs and at times fine moist rales. Pulmonary edema may develop. A rapid feeble pulse, diminution in blood pressure and other signs of shock may be present. As a rule the pulse is full, the pulse pressure high and the systolic blood pressure elevated. Blood flow is not retarded but may be accelerated. Venous pressure is normal.

At times the patients are delirious and may have convulsions or muscular twitchings. The pupils are usually contracted.

The *laboratory studies*: The blood picture is essentially normal. As a rule there is little if any dehydration and therefore the blood changes associated with dehydration are not present. The urine is concentrated, acid and contains albumin, casts and erythrocytes. The chloride excretion in the urine is reduced.

The blood proteins, hemoglobin, cell volume, erythrocyte count and hematocrit are normal or may be increased proportionately to a mild hemoconcentration sometimes present. The albumin and globulin ratio is not disturbed. The serum sodium and chloride are not usually changed. The chloride level is usually 98 to 100 m. eq. per liter (557 to 635 per cent sodium chloride). The sodium levels parallel the chlo-

ride. These values indicate that electrolyte changes in heat strokes are only coincidental if present.

The acid-base balance is changed to the acid side, the carbon dioxide content being definitely lowered. The high temperature, muscular activity with oxygen debt and lactic acid accumulation probably account in a large part for these changes.

GENERAL EFFECTS OF A TROPICAL ENVIRONMENT

The most common influence of a tropical climate is not to produce the definitely clear-cut syndromes described above. It is the mild influences which are of greatest importance to everyone, including the clinician. Invariably people who find themselves in a hot and especially humid environment for a prolonged period of time experience symptoms. These symptoms may be mild but are definite and are especially apt to be recognized upon entering a cool and comfortable environment. The symptoms consist of easy fatigue, inability to concentrate well, mental and physical inefficiency, decrease in appetite, insomnia, restlessness and irritability, sweating, and a general decrease in energy and interest in work. This is especially marked in colonists who enter the tropics from the temperate zones. The natives of the tropics seem to cope well with tropical climates. This general feeling of ill health and decreased efficiency is an important problem in public health and preventive medicine, as important, if not more important, as any concerned with communicable tropical diseases. The development of the tropics as a good place to live will be determined by the success with which the problem of heat transfer in the tropics is handled.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of heat cramps, heat exhaustion and heat stroke is best considered collectively, since they are so closely related mechanistically or certainly in their origin. They are all expressions of difficulties in the elimination of body heat. The identification of any of the three syndromes is not difficult if the patient is seen while experiencing his symptoms. All three entities are usually encountered in persons working hard in a hot environment. They are especially prone to development if the climate is tropical. In people working under such conditions, those who have the symptoms described above should be considered to suffer with a heat syndrome.

In heat cramps the severe pain and other symptoms make the diagnosis. At times the severe abdominal cramps may simulate an acute surgical abdominal disease. There is little confusion as a rule, for the history and examination and the response to salt and water therapy are diagnostic. In heat exhaustion there is the picture of circulatory collapse or shock and possibly the picture of heat cramps. At times the picture of heat stroke may develop. In heat exhaustion the body temperature is usually below normal, but it may be normal or elevated. Heat stroke is characterized by hyperpyrexia, dry hot skin and a state of marked mental confusion or coma.

Among the clinical states that may be confused in differential diagnosis are nocturnal cramps, cramps following violent exercise, gastric crises of tabes dorsalis, epilepsy, tetany, trichinosis, uremia, hysteria, malaria (especially cerebral malaria), and many other states. With a careful clinical work-up these states should not lead to confusion. Table 1 summarizes the significant differentiating characteristics of heat cramps, heat exhaustion and heat stroke.

TREATMENT

The *general treatment* is similar for the three syndromes and is therefore discussed as such first and then specific differences are indicated.

Prevention of the syndromes is more important in the management of the heat syndromes than the treatment of the syndromes *per se*. This aspect of the management is given serious consideration by the armies of the world and most industrial concerns. In the tropics measures to prevent the heat syndromes are not sufficiently emphasized. The old empirical and traditional methods employed by the natives of the tropics are essentially the basic principles still in use today.

The measures advocated to prevent heat

TABLE I.

DIFFERENTIATING CHARACTERISTICS OF HEAT CRAMPS, HEAT EXHAUSTION AND HEAT STROKE.

	Heat Cramps	Heat Exhaustion	Heat Stroke
Body temperature	Normal	Below normal usually, may be normal or elevated	Hyperpyrexia
Mechanism	NaCl lack	Circulatory collapse	Failure of the thermoregulating mechanism
Main symptom complex	Severe muscle cramps	Shock-like state	High fever, stupor, coma
Prognosis	Excellent	Good	Very poor — high mortality rate (10-80%)
Main therapeutic measures	Cool environment, rest, NaCl and water, morphine for pain	Cool environment, NaCl and water, rest	Ice tubbing, or wet sheets and fanning, rest
Incidence	Fairly common	Common	Relatively uncommon
Age	Any age, usually younger group	Any age, more common in senile subjects	Usually senile and debilitated individuals

cramps, heat exhaustion and heat stroke are the same for the three syndromes. This is to be expected since the mechanisms involved in each are similar. The state of health should be at best at all times, especially when one is exposed to a very hot environment. Frequent examinations with care of the teeth, avoidance of the use of alcoholic drinks even in relatively small amounts, and maintenance of a well-balanced diet should be advocated. The diet should not be excessive in calories and proteins and should contain the necessary vitamins. There should be a high sodium chloride intake, at least 15 grams a day. The fluid intake should be liberal, that is, sufficient to insure a urine volume of 1500 c. c. a day. The carbohydrate content of the diet should be great enough to supply most of the energy requirements.

Many measures have been advocated to insure sufficient *sodium chloride* intake to avoid disorders from the heat under exceptionally hot and environmental conditions in which strenuous exercise is necessary. For example, the U. S. Army advises the use of drinking water for its troops stationed in the tropics that contains sufficient sodium chloride to make a 0.1 per cent solution. To do this, one pound of sodium chloride is added to each 100 gallons of water, or 0.3 pounds to each 36 gallons Lyster bagful of water, or one-fourth teaspoonful to each quart (metal canteen) of water, or two 10 grain (0.6 gram) tablets of sodium chloride to each quart of water consumed, or one 10 grain (0.6 gram) tablet to each alternate glassful of water consumed.

Cool water should be drunk freely. If the urine volume of normal people is kept at 1500 c. c. a day one can be certain that the fluid intake is sufficient to meet the water lost in sweating, stools, respiration as well as other needs. In the climate of a tropical desert this often means drinking many quarts of water daily. Cool non-alcoholic drinks, such as fruit juices, are to be advised as they aid in the elimination of heat as well as supply water and vitamin requirements.

Since the main physiologic problem in the tropics is the elimination of heat, therefore the use of the *shade* of trees in the selection of sites for homes and places of business aid a great deal. Buildings should be built and roofed so as to render an environment as cool as possible. Shady verandahs, shuttered windows, well insulated roofs and walls are some of the measures that promote cooling. Ventilation should be at a maximum. The use of reflection by means of shiny metal roofs, white flies over tents aid in cooling a dwelling by reflecting the infra-red rays of the sun.

Resting: A good night's rest in a cool house with at least eight hours of sleep, is essential. A midday, after dinner, rest is of great value. In addition to physical rest, mental rest with frequent vacations are to

be emphasized. Not only does this promote efficiency, it aids in preventing the heat syndromes. People with conflicts and anxiety states or any of the psychoneuroses should be aided even if the advice of psychiatrists must be sought in extreme instances. People suffering with psychoneuroses cannot cope very well with the problem of heat elimination. The use of wholesome entertainment with recreation centers, especially with cool swimming pools, help. Methods to promote economic and family security are of paramount importance.

The *clothing* should not be tight and uncomfortable. It should be loose and light and should facilitate evaporation of sweat and additional heat loss by convection, radiation and conduction. The color of the clothing should be light in order to reflect light and heat. Shorts, shirts with short sleeves and low open necks are to be advised. Sun helmets which are light, well ventilated and lined with chromium foil are good.

Acclimatization is exceedingly important in facilitating one with coping with the tropical heat. It has been shown repeatedly in man and experimental animals that an animal cannot adjust very well to a sudden change to a tropical climate. People on first entering the tropics from temperate zones find it difficult to work well. These same people after a few weeks' sojourn in the tropics, work rather efficiently. The physiologic adjustments that are made with acclimatization are unknown in the main. It is well known that with acclimatization the capacity to sweat is increased manifold. The U. S. Army has long recognized the importance of acclimatization in the training of its troops for tropical campaigns by training them in the hot deserts.

Work should be planned so that the most strenuous work is done during the cooler hours of the day. The degree and duration of the work should be such as to keep heat production to a level that is within the limits of the physiologic mechanisms to eliminate the heat. After all, heat balance is an equilibrium between heat production and heat elimination which obeys the physical laws of heat transfer.

In industries the conditions under which men and women work should be comfortable. Not only should there be plenty of water and salt available, but the conditions of the immediate environment should be made cool by proper ventilation. The employees working near furnaces, and boilers and very hot machinery, should have an opportunity to rest in cooler places whenever they begin to experience the ill effects of the heat. By providing cheerful surroundings, frequent rests in cool places, plenty of cool water and plenty of salt, many men can work efficiently after acclimatization in extremely hot and humid environments.

THERAPY OF SPECIFIC NATURE

Heat Cramps: The treatment for heat cramps is empirical in the main, although certain definite curative procedures are indicated.

The patient should be removed to a comfortably cool environment as soon as possible. He should rest in bed. Many people recover without even resting but this is not to be recommended. Morphine, grain 1/4 to 1/2, should be administered immediately if the pains are severe. Avoidance of movement, external stimuli, and annoyances by observers and visitors are to be recommended as these factors are known to precipitate spasms. Hot packs over the muscles have been used, but are of relatively little benefit. The most important procedures consist of the administration of salt and water by mouth if the patient will take and retain it, or intravenously in severe acute cases. When the parenteral route is employed, 1000 c. c. or more (enough to keep the urine volume at 1500 c. c. a day) of physiologic salt solution is to be used intravenously. The usual precautions are to be followed as for all intravenous medication. When the salt is taken orally it is given as capsules or tablets, administering 15 to 20 grams daily and enough water to maintain a daily urine volume of 1500 c. c. Dextrose, orally or intravenously, may be used.

As a rule, rest in a cool place, salt and

water and morphine for the severe pain result in prompt and complete recovery. Usually a 1000 c. c. intravenous infusion of physiologic sodium chloride solution is sufficient to bring about satisfactory relief of the cramps.

The blood chloride levels are easy to determine and of value in the management of these patients. A simple rule to follow in otherwise normal patients is to give enough water to insure a urine volume of 1500 c. c. in 24 hours and an adequate urinary chloride excretion. The latter may be determined by taking 5 c. c. of urine, adding a few drops of 1 per cent silver nitrate solution, and than a drop or two of concentrated nitric acid. If the chloride concentration in the urine is normal a curdy precipitate forms, if it is low there is only a hazy white color change or no change at all. In applying these rules one must remember to take into consideration the presence of organic renal disease, heart failure, edema states or large urine volumes.

Heat Exhaustion: The treatment of this disease offers no difficulties. It is essential that the patient be removed from the hot environment to a comfortably cool one and allowed to rest. Usually that is sufficient. Sodium chloride and water administered as described above for heat cramps should be used. If there is collapse, concern is necessary only in senile or chronically diseased patients. Dextrose orally or intravenously may be used in liberal amounts. Intravenous medication should be employed with caution in patients with cardiovascular disease.

Heat Stroke: The treatment of this state offers a challenge to the physician. Early and satisfactory treatment is necessary as this disease is a medical emergency. Even with the best of treatment the mortality rate is high. The patient should be placed in a hospital immediately if possible. To reduce his body temperature he may be placed in wet sheets and fanned vigorously. When possible, it is better to place him in a tub of ice water with floating ice. The temperature of such a bath is about 35° F. When the body temperature reaches 102° F. the patient should be removed and the body temperature allowed to continue to fall normally. This continued drop is often claimed to be a cause of death in patients kept in the cold water too long. These patients should also be given sodium chloride and water as described for heat cramps. Intravenous fluids should be administered cautiously to the senile or debilitated patient. Dextrose, 5 per cent solution, may be given intravenously. The salt, water, and dextrose should be given orally whenever feasible. Lumbar punctures have been used to reduce the presence of the cerebrospinal fluid. Artificial respiration should be used freely if there is any need. Oxygen may be used. When the venous pressure is markedly elevated, venesection may be practiced. Recuperation should be permitted to occur slowly with the patient in bed. Many patients have overcome heat stroke but have died a few days later of heart failure or the like, upon getting up too quickly.

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CLINICO-PATHOLOGIC CONFERENCE

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NEW ORLEANS

The following clinico-pathologic conference was presented at Touro Infirmary on March 15, 1945.

Dr. F. C. Coleman: Dr. Stewart will read the clinical history.

This is the case of G. H., a white male, aged 64, who was admitted to the hospital on January 9, 1945, and died on January 18, 1945.

C. C.: Nausea and vomiting for three days.

P. I.: The patient felt perfectly well until three days ago when he suddenly developed a sharp pain in the upper midepigastrium. There seemed to be a considerable amount of gas on his stomach which aggravated his nausea. He has retained some water but no food. He has been constipated since the beginning of the present illness, whereas previously his bowel movements were regular every day. There has been no hematemesis, melena, jaundice, or clay-colored stools.

Review of Systems: *EENT*: Eye removed six years ago for cataract. *Lungs*: Morning cough productive of green phlegm since "flu" in 1918. *Heart*: Dyspneic on exertion for several years. No nocturnal dyspnea, no chest pain, and no cardiac irregularity. *G. I.*: Essentially negative until onset of present illness. *GU*: Essentially negative. *NM*: Marked weakness with tendency towards fainting.

Past History: Cholecystectomy 22 years ago. Has had ventral (incisional) hernia for 20 years.

Physical Examination: Blood pressure 130/90; pulse 70; respirations 18; temperature 98.6. *Head*: Negative. *Eyes*: Artificial eye in left orbit. *Throat*: Negative. *Lungs*: Clear to auscultation and percussion (examination unsatisfactory due to noises about the ward). *Heart*: The heart is enlarged one cm. to the left. No murmurs were heard. Extrasystoles were present at about every 10th impulse. *Abdomen*: The abdomen is flat and has a bulging incisional hernia in the midline, the ring of which measures 3 to 4 cm. in diameter. There is an indefinite mass in the midepigastrium which is slightly tender.

Laboratory Data: On January 10, 1945, urinalysis revealed an acid urine with a specific gravity of 1.024; trace of sugar; many bacteria and occasional pus cells. Repeated blood chemistries revealed a total NPN which varied from 50 mg./100 c. c. on January 10, 1945, to 34 mg./100 c. c. on January 15, 1945; dextrose level was 80 mg./100 c. c. and sodium chloride 458 mg. per cent. CO_2 combining power 52.2 volumes per cent. Serology done on January 10, 1945, was negative. A hemogram done on January 17, 1945, revealed a total RBC of 3.5 million with 72 per cent hemoglobin, a total white blood count of 8,000 with 72 per cent neutrophils, 14 per cent lymphocytes and 4 monocytes.

Electrocardiogram taken on January 16, 1945, revealed a definitely abnormal tracing with possible acute anterior occlusion.

Clinical Course: The patient was admitted on January 9, 1945, and at that time was thought to have a high intestinal obstruction. He had pain in his midepigastrium with nausea and vomiting. The nausea and vomiting subsided rapidly and the patient began to feel well. By January 13, 1945, he was permitted to be up and about in a wheel chair. On January 14, 1945, his temperature rose to 102 degrees. He had no complaints except the inability to sleep at night for which a sedative was prescribed. When he first was admitted enemas produced good results. On January 17 he was put on complete bed rest after electrocardiographic report was received. On January 18 the patient's heart rhythm was noted to be irregular with skipped beats and he complained of a severe pain over the sternum. On the afternoon the patient suddenly took two gasps, stopped breathing and fell across the bed. He was cyanotic. Auscultation of the heart revealed a rapid rate of over 300 which was thought to be ventricular fibrillation. This lasted for 30 seconds. The patient expired at 4:55 p. m.

Dr. George Stewart: I saw this patient when sent to us by one of the staff men in the back ward of surgery. We anticipated removing the incisional hernia; however afterward he complained of pain in heart, dyspnea and cardiac symptoms. With consultation we obtained an electrocardiogram which was abnormal in many respects and so postponed surgery until further recommendation from medicine.

Dr. Coleman: Are there any questions about the history?

Dr. J. D. Rives: What about the epigastric mass mentioned in the original examination but not mentioned again? What became of it?

Dr. Stewart: It remained there and stayed practically the same. We thought we could feel some hard material in the intestine but since bowels were moving perfectly and there were no more epigastric symptoms or vomiting or nausea, we decided that it would be all right until we found out more from medicine.

Dr. Philip M. Tiller: I would like to know how long a period of time elapsed from the time he gasped until he was actually dead.

Dr. Stewart: From time seen about thirty seconds.

Dr. Tiller: How long before that?

Dr. Stewart: Five minutes. Dr. Levine was on the ward at the time he had the attack. He was seen five minutes after the first episode.

Dr. Coleman: Dr. Philip Tiller will give the clinical discussion.

Dr. Tiller: We have a story of a 64 year old white man who suddenly developed epigastric pain, and who died, nine days after being admitted to the hospital, in what was apparently an acute circulatory affair. On January 6 he suddenly had epigastric pain. I presume it was severe. It was stated to be sharp, but it is not stated whether or not it was continuous pain. He became nauseated shortly after that and for three days retained no food, little water, and had no bowel movement. His blood pressure was 130/90 when first seen. Examination of the heart and lungs, even though it was unsatisfactory to the intern, indicates pretty well that he was in no circulatory embarrassment at the time; that he had no heart failure. The abdomen was flat and apparently the hernia was not the cause of his pain. The indefinite epigastric mass I think I will dismiss as a red herring. The epigastrium is almost the natural habitat of phantom masses, and I cannot make it fit in with my ideas anyway.

His urine concentrated to 1.025; the NPN 50. I think those two things indicate fairly good kidney function and an increase in NPN due to dehydration. The fact that NPN was normal shortly after that helps us in that idea. The other laboratory findings were essentially normal.

He had a mild anemia. One thing that is not mentioned is sedimentation rate. On January 16 it was 95 millimeters per hour, Westergram method, so he definitely had an increased sedimentation rate.

Four days after the onset he was well enough to be in a wheel chair. The nausea and vomiting disappeared and he apparently was strong enough, and thought well enough to be up. A cardiogram taken on the sixteenth, seven days after admission, is certainly very interesting. There are low R waves in lead I, in some places no R wave at all. The S-T segment is definitely elevated in lead I and depressed in lead III. In the precordial leads a Q wave is present in CF I, a deep Q is present in CF3 and still a Q wave present in CF5. Again in CF5 a Q wave is present that is almost the same length as the R wave in that lead. The ST segment is elevated in CF3 and CF5, is slightly elevated in CF1.

I think we can make a diagnosis of coronary occlusion on the basis of the cardiogram, but cannot say flatly that it occurred on the date that his symptoms started—on the sixth of January—because he may have had an old anterior infarct giving him his Q waves in CF1, 3 and 5, and he may have had a transient anterior ischemia causing temporary elevating of the ST segments in lead I and depression in lead III with elevation in ST segment in the precordial lead. However, for all practical purposes we can assume that the cardiogram establishes a diagnosis of acute coronary infarction in the anterior wall of the left ventricle probably in the region of the septum, and extending far laterally; a very extensive infarct.

I worked a little backward on this case because I made the diagnosis before discussing other possibilities. We are discussing this on the basis of hindsight anyway, so I guess that is probably all right. There are other things we certainly have to consider as possibilities.

If he had an intestinal obstruction as was suggested, that obstruction was transient because he certainly had no obstruction on the thirteenth of January, four days later; four days after admission. Also, things that will cause transient intestinal obstruction in a man his age are not very common. A volvulus occasionally will do it, but this is not too common a diagnosis. A volvulus that will occur and cause symptoms of obstruction and then disappear is less com-

mon. Carcinoma that causes sudden obstruction because of edema in the bowel around the area of carcinoma would probably not cause such sudden severe pain and I cannot think where the carcinoma would be, unless in the colon, that would cause obstruction, and you would expect the symptoms to be progressive over a long period of time. The gallbladder was out. It is possible—theoretically possible—that one may have colic from stones in the hepatic radicles or common duct but that gallbladder was out twenty years ago, so such an occurrence would be unusual. A perforation of a peptic ulcer in the lesser sac is a very confusing condition. The tenderness in the midepigastrium might incline us to that diagnosis just a little bit, but I would expect a higher white count than he had. Besides we have no previous history indicating peptic ulcer. Mesenteric thrombosis is again a confusing diagnosis to make. I do not think it occurred in this man because he had no previous history of vascular disturbance, it is not stated that he had bloody stools afterward, and he recovered rapidly. If it actually occurred, the thrombosis must have been small. There was no tenderness in the right lower quadrant so I guess it was not appendicitis. A pulmonary infarct is possible with a sudden onset of epigastric pain, but there were no pulmonary symptoms following that. I think I will stick to the diagnosis of coronary occlusion that began on the sixth.

There are one or two things of interest there. Why did he vomit so much afterwards? Nausea and vomiting are not too uncommon with coronary occlusion; possibly related to pain or circulatory disturbance that occurs at the time. People with coronary occlusion usually have pain severe enough to get morphine. He may have had morphine and that may have precipitated vomiting. When a patient vomits enough to become dehydrated there is almost a vicious cycle set up; dehydration continues the nausea and vomiting, and it is necessary to give fluid by vein to break the cycle. I think those things are not incompatible therefore with the diagnosis of coronary occlusion.

Now, we come to the final episode. On the eighteenth his heart rhythm was noted to be irregular. Then he had severe pain located in the region of the sternum. Following that—we do not know how long—he suddenly gasped, fell across the bed and within a period of about five minutes died. There are several things that we have to consider as possible mechanisms in this man's death. Did he have another coronary occlusion with infarction that began with pain earlier in the day and did his death result from a sudden ventricular fibrillation? Secondly, did he rupture the myocardium in the area of infarct? Thirdly, did he have cerebral embolus? He took five minutes to die. Usually with a ruptured myocardium death is almost instantaneous, sometimes lasts a time. Ventricular fibrillation usually will kill a person in thirty seconds or less. With cerebral infarct the patient may live for some time afterward. If he had a cerebral embolus, of course, it would have come from a mural thrombus in the left ventricle. It is stated that when seen his heart rate was over 300. You could safely say over 200. When you get to a heart rate that high it is a little difficult to count and it is a little difficult to be sure how fast the rate was. We are not told whether regular or not. If going over 200 I do not think I could tell. I do not think the arryhythmia was the precipitating cause of death; either myocardial rupture or cerebral embolus killed the patient, and the rapid cardiac rate heard by the intern who saw him was probably a terminal event.

Dr. Coleman: Thank you Dr. Tiller. Are there any comments from the clinical aspect of this case?

Dr. Willard Wirth: I would like to comment on the electrocardiogram. My interpretation, based upon the findings which Dr. Tiller enumerated, was that is was definitely abnormal and, as I wrote on the electrocardiogram, probable acute anterior occlusion and I also suggested that the electrocardiogram be repeated. I put my inter-

pretation that way for reasons that Dr. Tiller has hinted at. Sometimes these changes are transitory, associated with some transitory coronary ischemia and repeated electrocardiograms will show reversal of these changes in that case, or in a case of true occlusion with myocardial infarct repeated electrocardiograms would note progressive changes and allow a more emphatic statement that it was a definite acute occlusion with myocardial infarct. This electrocardiogram is quite characteristic, however, and would be entirely compatible with an acute coronary occlusion. By acute I would say that we were not able definitely to date the acute episode from a single electrocardiogram but would supose that it was an accident that occurred within a week or maybe a little longer rather than many weeks or a month or so.

In addition this electrocardiogram showed frequent premature contractions which occurred in a fashion to produce intervals of pulsus bigeminus or coupling so at the time the electrocardiogram was taken this man had an irregular rhythm.

Dr. Coleman: Any other comments?

Dr. John H. Musser: Of course the man had ventricular tachycardia and not fibrillation. You could not count the pulse or hear the heart sounds in ventricular fibrillation. Tachycardia so often precedes fibrillation that that is one of the reasons we give quinidine to these patients.

Dr. Coleman: Are there any other comments?

Dr. I. L. Robbins: I think in a differential diagnosis, such as this, especially with severe epigastric pain, dissecting aneurysm should be considered.

Dr. Coleman: At postmortem the body weighed 100 pounds and measured 5 feet 8 inches in length. An artificial eye was present in the left orbit. Postmortem lividity was marked over the dependent portions of the head and neck. A ventral hernia was present in which was encarcerated the hepatic flexure of the colon and the right half of the transverse colon. The encarcerated bowel was reddish brown, whereas the remainder of the colon was yellowish gray. No excess fluid was present in the peritoneal cavity. No excess fluid was present in the pleural cavities. The liver weighed 1200 grams as against the normal 1500 grams. Mild nutmegging was present on the cut surface. The gallbladder was absent. The spleen weighed 100 grams as against a normal of 160 grams. The kidneys were normal in size but the outer surfaces were pitted. The hepatic flexure of the colon and the right half of the transverse colon were attached to the walls of the ventral hernia by firm fibrous adhesions. No obstruction was present. The brain grossly and microscopically showed no lesions. No lesions of the pancreas or adrenals were present. The heart weighed 325 grams. The pericardial sac was larger than normal and on external examination, had a bluish red color. It was filled with blood clot of approximately 60 c. c. in amount. This blood clot was readily removed from the anterior surface of the heart.

Three centimeters above the apex of the heart on the anterior surface there was a ragged tear in the heart muscle, measuring 1 cm. in length and 2 mm. in width. A transverse incision was made through the jagged opening and it was found to communicate with the apical portion of the left ventricle. Parallel transverse incisions were made throughout the anterior surface of the heart and the interventricular septum. A myocardial infarct was present, involving the entire anterior wall of the left ventricle and the anterior two-thirds of the interventricular septum. The infarcted muscle was soft, friable, and dull yellowish gray. This yellowish gray color was replaced by a dark reddish brown color, however, for 2 mm. to each side of the tear in the myocardium. The infarction also at this point extended down to the endocardial surface whereas throughout the remainder of its extent it did not involve the myocardium for a distance of 2 to 3 mm. below the endocardial surface. It did involve, however, the muscle completely out to the pericardium. The coronary arteries were dis-

sected out and a thrombus 1.5 cm. in length was present in the anterior descending branch of the left coronary artery beginning 5 mm. below the origin of this vessel. This thrombus was friable and dark brown in color.

Fig. 1. Photograph of apex of heart with left ventricular chamber exposed. Note area of hemorrhage and necrosis in anterior wall.

Fig. 2. Photograph of heart with anterior descending branch of left coronary artery sectioned. Note thrombus occluding the vessel.

This 35 mm. Kodachrome photograph of the heart is taken from the anterior surface demonstrating the tear in the myocardium. This 35 mm. Kodachrome photograph is taken also from the anterior surface and shows a close-up view. Note the blood clot protruding from the tear. In this photograph, the left coronary artery has been sectioned and the ends everted to demonstrate the thrombus. This low power magnification photograph of the myocardium is taken from the infarcted portion of the anterior wall of the left ventricle near the epicardial surface. The muscle fibers which remain are pale; their striations are lost and their nuclei are gone. Fragmentation is present and a smudgy type of necrosis is noted in which degenerating polymorphonuclear leukocytes are present. Some of these are noted also between the infarcted muscle fibers. This low power magnification photograph taken from the anterior wall of the left ventricle in the infarcted area shows granulation tissue growing from the epicardial surface inwards into the infarcted area. The granulation tissue is loose, moderately vascular, and a few

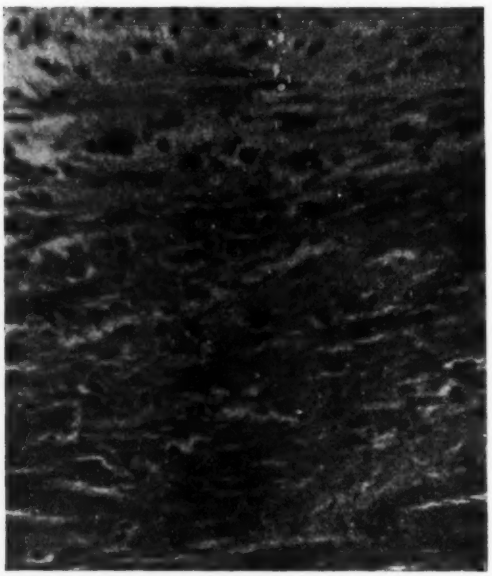

Fig. 3. Micrograph of infarcted myocardium. Note smudgy type of necrosis.

polymorphonuclear leukocytes are present. Macrophages also are present, some of which contain yellowish brown hemosiderin pigment. These macrophages are important in removing the dead muscle tissue. A few muscle fibers remain which have survived. These are surrounded by granulation tissues.

In review, the anatomic findings are:

1. Cardiac tamponade.
2. Myocardial infarction of the anterior wall of the left ventricle, and anterior two-thirds of the interventricular septum.
3. Rupture of the anterior wall of the left ventricle.
4. Thrombosis of the anterior descending branch of the left coronary artery.
5. Chronic passive congestion of the lungs, liver and spleen.
6. Ventral hernia with encarceration of hepatic flexure of colon and right half of transverse colon.
7. Chronic pyelonephritis.

Histologically, we are able to determine the approximate age of myocardial infarction. The sections in this case place the time of infarction at approximately the same time as the onset of symptoms. Nausea and vomiting without precordial pain are sometimes seen in coronary occlusion as is well demonstrated in this case. The occlusion was complete and was located in the anterior descending branch of the left coronary artery. Anastomatic circulation was poor and massive infarction of the anterior wall of the left ventricle and the anterior two-thirds of the interventricular septum resulted. A few hours before death, the first precordial pain was present. Ten days after infarction had occurred, the infarcted area had become soft enough as result of necrosis and phagocytosis of dead muscle tissue so that rupture took place. Cardiac tamponade occurred and the patient died within five minutes. The ventral hernia with encarcerated bowel was merely an incidental finding. The tumor mentioned in the history as being present in the hernia consisted of encarcerated bowel.

Next slide: *Coronory Occlusion*

1. Forty per cent occur in males over 55 years.
2. Eleven per cent of hearts studied (2.898 cases) showed evidence of coronary occlusion. The arteries were injected with a lead-agar preparation; the hearts were dissected in a special fashion so that the coronary arteries lay in one plane and x-rays taken in order to localize the occlusions. The occlusions were then carefully dissected out.
3. Two-thirds of occlusions are less than five millimeters in length.
4. Anterior descending branch of left coronary most frequently involved but circumflex branch of left coronary and right coronary also frequently involved.
5. Sixty-nine per cent of occlusions occur in proximal four centimeters of coronary vessels.
6. There is no relationship between branching of coronary vessels and localization of occlusions. This indicates that some of the older theories as to the method of branching, whether at right angles or an acute angle, have nothing to do with production of thrombosis at that point.

Changes in the myocardium following infarction:

1. Gross changes:
   a. Earliest change—Involved area pale and drier than normal with occasional focal areas or hemorrhage.
   b. Four days—Fine yellow line around periphery progressing in extent (leukocytic infiltration).
   c. Ten days—Reddish purple zone around periphery (new capillaries) and removal of dead muscle begins.
   d. Four weeks—granulation tissue with small islands of necrotic tissue remaining.
   e. Three months—Shrunken white fibrous scar is all that remains.
2. Microscopic changes:
   a. Four to six hours after infarc-

tion—Muscle fibers appear hyaline and striations indistinct. Infarction as a rule does not involve three to four millimeters below endocardium (Thebesian veins).

b. Twenty-four hours—Polymorphonuclear leukocytic infiltration begins and continues through first four days.
c. Fourth day—Capillaries begin to grow in. Begins peripherally pericardial surface) and extends toward the center.
d. Sixth day — Polymorphonuclear leukocytes are degenerating. Are gone by fourteenth day.
e. Tenth day—Removal of dead muscle fibers by mononuclear cells beginning at periphery.
f. Twelfth day—Collagen fibers formed; prominent at three weeks.
g. Three months—Marked scar tissue formation.
h. Endocardial thrombi may appear by fifth day and are organized by sixteenth day.

Myocardial infarction with cardiac rupture:

1. Eight and three-tenths per cent of fatal myocardial infarcts show rupture.
2. Age incidence of infarction with rupture parallels that of infarction without rupture.
3. Rupture most frequent in anterior wall of the left ventricle. (Anterior descending branch of left coronary most frequently involved.)
4. Rupture most likely to occur in small infarcts. (Square area of five centimeters or less).
5. Rupture usually occurs between third
6. Proportion of rupture highest in cases with small hearts and hypertension persisting after infarction. It is interesting to note that the heart in this case was normal in size.
7. If scarring is present in myocardium, rupture only one-fourth as likely as in unscarred hearts. This is thought to be due to the presence of collateral circulation and supporting action of scar tissue. For practical purposes, collateral circulation is non-existent in hearts which have not undergone infarction.
8. Cases with rupture show microscopically.
   a. Smudgy type of necrosis with heavy infiltration of leukocytes. (Enzymatic action of leukocytes).
   b. Hemorrhage.
9. Patients with interventricular rupture live an average of 2.25 days as contrasted with those who have a rupture into the pericardial sac. These die suddenly.

Are there any additional comments?

Dr. Allan Eustis: There is just one comment I would like to make. If you noticed, this man was up in a roller-chair four days after initial onset. For twenty years I have made patients keep in bed for two weeks after suspected coronary occlusion whether clinically well or not. It is far better to keep in bed than have an end like this where you are liable to be open to criticism.

Dr. Coleman: Any other comment?

Dr. J. R. Schenken: I would like to make one comment with regard to the survival time following cerebral vascular accidents of any type. Among those patients who die following thrombosis, embolism or spontaneous massive cerebral hemorrhage, the vast majority live more than two hours. I have never seen a patient who died in less than eight minutes following the onset of the disease. If death occurs within a few minutes it is almost certain that the cause of death is not due to some spontaneous cerebral vascular accident.

NEW ORLEANS
Medical and Surgical Journal

Established 1844

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1430 Tulane Avenue

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THE JOURNAL does not hold itself responsible for statements made by any contributor.

LAY CONTROL OF MEDICAL PRACTICE

The monthly news letter from the American College of Radiology is interesting and thought provocative. In the May number of this publication there is a short presentation entitled "Medical Societies Tangle With Blue Cross." The gist of this presentation has to do with the Blue Cross proposing to contract with doctors to furnish medical practice to subscribers. The rightful contention is, should this procedure become widespread or even become started, that the hospitals would no longer be institutions for the care of the sick but as they could contract for the sale of medical services they would engage in the practice of medicine. As the officers of the Medical Society of Wisconsin say "the issue here is whether medicine is to be practiced *in* a hospital or *by* a hospital."

The reasons that the hospitals feel that they should be entitled to contract for medical services is because a beginning has already been made in doing this very thing. The American College of Radiology points out that under the Blue Cross plan in many places radiology and pathology are included in hospital benefits and that consequently if one or two specialties of medicine are to be included among hospital benefits the entering wedge has been made whereby other branches of medicine will be called upon to act under the jurisdiction of a hospital board. Such a procedure is pernicious. The controversy which is now raging in Pennsylvania and Wisconsin illustrates well indeed that once there appears a crack in the door it will not be long before the door is wide open to permit the entrance of all kinds of schemes for medical practice about which the doctor has little to say.

THE NEED FOR NURSES

There still exists, according to the Army authorities, a rather dire lack of nurses. While the Army Nurse Corps has at the present moment 51,000 nurses, they claim that they need 9,000 more. The Veterans Administration needs a 50 per cent increase in the number of nurses who are now working in that organization. It is claimed also that essential nurses are needed in industries, national and state public health services, in schools and other places where a nurse is deemed essential to the proper care of war casualties and to fill civilian needs.

It might be pointed out that there are slightly under 300,000 nurses in the country at the present time. There are also many inactive or retired nurses. In addition to the available nurses, slightly under 200,000 volunteer nurses aides have completed their

training and some 50,000 of these young women are now serving in hospitals.

While there may be a need for additional women to enter the nursing profession and there may be a lack of nurses for essential nursing positions we cannot help but feeling that many nurses are filling positions which certainly do not require women who are trained in the profession of nursing. It seems not so remarkable that with 62,000 nurses in the armed services that there is a so-called shortage of nurses. Looking back to the days of the depression, nurses literally, unless unusually qualified, were starving. There was a tremendous surplus of nurses at that time. When and if the boom times of war quiet down again, there will be a difficulty in finding positions and jobs for the large number of women who, under the stimulus of governmental subsidy, are being trained in nursing schools. Many of these nurses-to-be will find it will be impossible to make a living in their profession. We are skeptical about the so-called need of thousands of more nurses and feel that the exaggeratedly large number of nurses now being added each year to the profession will make it difficult if not impossible for many of these women to make a living in their profession.

PENICILLIN IN ASTHMA

At times one of the most intractable and difficult conditions to treat is bronchial asthma. Fortunately in the great majority of cases it is possible to determine the allergen to which the patient is sensitive and by excluding contact with that allergen the patient's asthma may be entirely relieved. When this is not possible, the allergists in many instances are able to desensitize the patient so that his reaction to the offending antigen or antigens which cause trouble is reduced to a minimum or entirely deleted. There are, however, a certain number of patients in whom treatment is notably ineffective. This group of patients usually suffer from intrinsic asthma, that is, the type of asthma which, according to Rackemann, "the cause of the trouble lies within the body." In extrinsic asthma the offending cause can usually be determined but in the intrinsic type there is some unknown factor, the "x factor," which is responsible for the symptoms but what this antigen is cannot be detected by any diagnostic procedure.

Penicillin has been used for almost every condition, disease or disorder to which man is heir, even cancer. It has been found to be unexpectedly effective in many conditions which it is hard to believe the drug would benefit. In intrinsic asthma, however, it is possible that the x factor may be bacterial. That this supposition may be true is confirmed by a paper by Leopold and Cooke* in which they detail two case reports in which penicillin was employed with rather remarkable results. In both these two treated individuals the asthma continued despite every type of treatment over a long period of time. Penicillin was then administered and both of the two patients showed a remarkable improvement. These two physicians, both men of reputation and standing, also have under treatment twenty-five other patients with intractable asthma. They say it is too early to comment upon the results of the treatment of these patients but that it is obvious penicillin is not a panacea for all cases of asthma due to infection, as some of the patients have failed to show improvement with penicillin therapy. Be that as it may, the report of these two observers would indicate the use of penicillin in intractable cases of asthma when other forms of therapy have proved ineffective.

*Leopold, S. S., and Cooke, R. A.: Penicillin in the treatment of intractable bronchial asthma. A preliminary report, Am. J. Med. Sci., 209:784, 1945.

ORGANIZATION SECTION

The Executive Committee dedicates this page to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

THE NEW WAGNER BILL
(S. 1050)

The above bill was introduced in the Senate on May 24, and referred to the Senate Committee on Finance, of which Senator George, of Georgia, is chairman. There is a companion bill introduced in the House by Mr. Dingell, known as H.R. 3293. There has been a great amount of literature from various medical circles, attempting to analyze the provisions of these comprehensive amendments to the Social Security Act. The new bill is 185 pages and contains therein all the different propositions offered for social security. Following is a brief summary of the provisions of the bill:

Section 1—Short Title: Social Security Amendments of 1945.

Sections 2 and 3 provide for grants and loans to states for construction of hospital and health centers.

Sections 4 and 5 provide for federal grants to states for expansion of public health, maternal and child health, and welfare services.

Section 6 provides for federal grants to states for an expanded comprehensive public assistance program.

Sections 7 and 8 provide for continued federal operation of the United States Employment Service.

Section 9 establishes a National Social Insurance System, in eight parts:

A. *Prepaid Medical Care Insurance*, including services of physicians, surgeons, dentists, nurses, use of laboratory and x-ray equipment, and hospitalization, up to 60 days a year or possibly longer; covers all employees and dependents except self-employed.

B. *Unemployment and Temporary Disability Insurance*—up to $30 a week for 26 weeks or longer. Covers employees, except self-employed.

C. *Retirement, Survivors' and Extended Disability Insurance Benefits* — expands present OASI system as to benefit scale to a maximum of $120 per month and a minimum of $20 per month. Retirement age for women reduced to 60 years. Total disability benefits on same scale. Lump sum death benefits to spouse.

D. *National Social Insurance Trust Fund* established.

E. *Credit for Military Services* based on $160 wages for each month of military service.

F. *Extends Coverage* to about 15 million additional persons.

G. *Contribution Rates* established at 4% on first $3600 for earnings for employee, same for employer; 5% of value of services up to $3600 for self-employed.

H. *General Provisions.*

In the June 2 issue of the Journal of the A. M. A., page 364, there appears a most interesting and instructive editorial. On page 369 of the same issue is an analysis by the A. M. A. Bureau of Legal Medicine on the compulsory medical insurance features of the bill.

It may be of interest to you to know, from the statements made by Senator Wagner, the groups which are supporting him in the *rearrangement* of this bill in comparison with his federalization bill of the 78th Congress, known as Senate Bill 1161. He states that he has consulted the American Federation of Labor, the Congress of Industrial Organizations, the Physicians Forum, the Committee of Physicians for the Improvement of Medical Care (original 400) and the National Lawyers Guild, among other organizations and has secured approval of these groups for the changes made. Space will not permit an extensive analysis of the policies of these groups; however, we do know that, with the exception of the Na-

tional Lawyers Guild which we have recently learned is an organization not approved by the American Bar Association, they supported the first Wagner bill. Senator Wagner did not, however, take into his confidence any representative group of the A. M. A.

The above information is being brought to your attention for the specific purpose of asking that you write to your congressman or senator, secure a copy of this bill, read it, make up your mind about it, and write a letter to him expressing your views concerning same. It is urgent that you attend to this important detail now.

In the June 9 issue of the Journal of the A. M. A. there is a letter from Senator Wagner explaining his attitude toward the medical profession and stating specifically that he is opposed to socialized or federalized medicine. This is rather paradoxical in view of the various provisions of the bill which permit the Surgeon General of the United States Public Health Service to direct and supervise the practice of medicine and its various allied branches. One who has studied this subject seriously cannot help but realize the past and present neglect in preventive medicine activity. Some seem to centralize their ideas on the thought that the existing system of medical practice is inadequate and must be changed. The causative factors of illness, such as improper nourishment, vicious habits, ignorance, heredity, and modes of living, dependent upon poverty or illiteracy, are given little attention by these propagandists who propose a new social order for our medical practice.

The best information we have is that the hearings on the Wagner-Murray-Dingell bill will not be held for some time. The 1945 edition is twice as long as the 1943 volume. It is indicated, first, that this will not be rushed through Congress; second, that plenty of time for analysis and study by local medical groups will be given; and third, that the up-to-date press comment, generally, has not been overenthusiastic about the bill.

The bill includes almost entirely the contents of Senate Bill 191, known as the Hospitalization Bill. Fortunately, or otherwise, for the proponents of Senate Bill 1050, Senate Bill 191 has been approved, in substance, by the A. M. A., the American Hospital Association and the American College of Surgeons.

It is also very desirable that after you have given this critical subject your serious attention, you communicate with the executive officers of the State Society, giving them your opinion and information as to any action taken by you in opposition to same. Our Committee on Congressional Legislation is studying the provisions of this bill and any communications on the subject addressed to 1430 Tulane Avenue, New Orleans, with constructive suggestions, will be greatly appreciated.

The Veterans Administration is still under fire. All kinds of bills, investigations and allegations have been offered in Congress, by the lay press and officers of the U. S. government. It appears that the administration will be completely reorganized in an attempt to supply medical and hospital facilities to meet the growing demands of returning veterans.

Information appears in the press, as well as in medical circles, that some of the medical officers returning from Europe since V-E Day are being assigned to permanent positions in the Veterans Administration. This is against the wishes of the medical officers and protests have been made by the Committee on Postwar Medical Service of the A. M. A. to the Surgeon Generals of the Army and Navy objecting to such a procedure. It was hoped that some 9,000 doctors would be returned from Europe and discharged with the 1,800,000 military personnel. These doctors are acutely needed in certain areas of our state where the civilian doctors have been under a tremendous strain during the war.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

CALENDAR OF MEETINGS

July 4. Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.
July 5. Clinico-pathologic Conference, Touro Infirmary, 12 noon.
July 9. Scientific and Second Quarterly Executive Meeting, Orleans Parish Medical Society, 8 p. m.
July 18. Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.
July 19. Clinico-pathologic Conference, Touro Infirmary, 12 noon.
July 25. Catholic Physicians' Guild, 8 p. m.
July 26. DePaul Sanitarium Staff, 8 p. m.
July 27. L. S. U. Faculty Club, 8 p. m.
New Orleans Hospital Dispensary for Women and Children Staff, 8 p. m.

The June scientific meeting of the Orleans Parish Medical Society, which was held on June 11, was presented by members of the faculty of the Louisiana State Medical Center. Drs. Jesse Colley and Wallace Sako presented a paper on Pneumococcus Meningitis in New Born Infants; Mr. D. Postell read a paper on Early History of Tuberculosis in Louisiana; and Dr. C. A. Stewart read a paper on Congenital Absence of the Lung.

NEWS ITEMS

The following members of this Society addressed the annual meeting of the Louisiana Medical, Dental and Pharmaceutical Association held May 15-16 at Flint-Goodridge Hospital: Drs. R. E. Arnell, O. W. Bethea, Val Fuchs, John H. Musser, Paul T. Talbot, Sidney L. Tiblier, Waldo A. Treuting and Theo. A. Watters.

Dr. Sidney Jacobs spoke on Immigration Into Palestine at a recent meeting of the Tulane University-Newcomb College War Forum.

At a recent meeting of the Louisiana Association of Pathologists Dr. Norman T. Conant, associate professor of bacteriology, Duke University, spoke on Microbe Diseases in Man. Dr. J. W. Davenport, Jr., presented a paper on Practical Aspects of Rh Blood Type.

The following officers for the ensuing year were elected: Dr. William H. Harris, president; Dr. C. H. Binford, vice-president, and Dr. Emma S. Moss, secretary-treasurer. Dr. John R. Schenken, the retiring president, will serve as a member of the executive committee.

Dr. P. Jorda Kahle was recently elected an honorary member of the L. S. U. Undergraduate Honorary Scholastic Medical Society, the Circle, in recognition of his original work in the use of stilbestrol in the treatment of carcinoma of the prostate. Dr. Kahle addressed the Society at the annual Circle banquet; his subject was, Review of Forty Years of Progress in the Field of Urology.

At the annual meeting of the board of directors of the Tuberculosis Association of New Orleans held May 3 in the Hutchinson Memorial Auditorium the following members of this Society were elected to office: Dr. John H. Musser, re-elected president; Drs. Julius Lane Wilson, William A. Sodeman, Chester A. Stewart, Edwin L. Zander, John M. Whitney, Louis A. Monte and Maurice Campagna, members of the board.

Dr. Howard Mahorner was chairman of the wartime graduate medical meeting held at the United States Naval Hospital on May 23. At this meeting Dr. Philip M. Tiller, Jr., gave a dry clinic; Dr. Leon Menville spoke on roentgenology in diagnosis of bone tumor; Dr. Alton Ochsner spoke on neurovascular lesions, and Dr. Theo. Simon spoke on low back pain.

The following members of the Society attended a recent discussion meeting under the auspices of the Medical Advisory Committee of the Office of Scientific Research and Development in New York City: Drs. George Burch, B. I. Burns, Alton Ochsner and John R. Schenken.

Dr. Emma Moss was recently promoted from lieutenant to captain in the Civil Air Patrol.

Dr. John H. Musser was recently elected a member of the National Board of Medical Examiners.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria

Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

SECOND DISTRICT MEDICAL SOCIETY

At a recent meeting of the Second District Medical Society held in LaPlace, the following officers were elected for the year 1945: President, Dr. J. W. Atkinson, Gretna; Secretary-Treasurer, Dr. Robert A. Katz, New Orleans.

EIGHTH DISTRICT MEDICAL SOCIETY

A meeting of the Eighth District Medical Society was held in Alexandria on June 4. Dr. Edgar Hull, professor of medicine of L. S. U., presented a paper entitled "Errors in Academic and Practical Diagnosis." The Secretary of the State Society, Dr. P. T. Talbot, was present and made a talk on modern trends of medicine, especially in relation to congressional action in Washington.

Doctors in Louisiana, as well as throughout the country, should be interested to know that a bill proposing compulsory health insurance which was submitted to the California Legislature was recently defeated by a large majority.

SOUTHERN MEDICAL SOCIETY

It is with regret that we announce the death of Dr. Edgar G. Ballenger, President of the Southern Medical Association.

Dr. Ballenger died on the first of June as a result of a fall from the seventh floor of an Atlanta hotel. He will be succeeded by Dr. E. Vernon Mastin, who was elected Vice-President of the Southern Medical Association at the last meeting.

REGULATIONS FOR USE OF DIATHERMY MACHINES

Following is copy of letter which was forwarded to the office of the State Society by Senator John Overton subsequent to correspondence with him concerning information on this subject. Copy of the report mentioned in this communication is on file in the office of the Secretary-Treasurer and may be reviewed by any member who desires further data in this regard.

FEDERAL COMMUNICATIONS COMMISSION
WASHINGTON, D. C.

May 26, 1945

Dear Sir:

This is with reference to our previous correspondence concerning the proposal of the Commission to allocate certain frequency bands in the radio spectrum for the use of medical equipment such as diathermy apparatus. The Commission's proposal in this regard was set forth in its report of January 15, 1945, and was later the subject of oral argument and briefs filed by interested parties. After having carefully reviewed all available data including that presented in briefs and at oral argument the Commission on May 17, 1945, announced a final allocation of frequencies for medical use and on May 25, 1945, issued a report discussing the basis for that allocation. In view of your interest in this matter we are enclosing a copy of that report for your information.

You will note that the final allocation for medical use consists of the following three frequency bands:

Center Frequency Megacycles	Band Width (% of Center Frequency)	Band Width (Kilocycles)
13.66	±.05%	15
27.32	±.5%	270
40.98	±.05%	40

With one change this final allocation is the same as that originally proposed by the Commission in its report of January 15, 1945. The change consists of an expansion of the 27 megacycle band from an originally proposed width of plus or minus .05 per cent of the center frequency to a width of plus or minus .5 per cent of that frequency. In terms of kilocycles this has involved an increase in the size of the band from a proposed width of approximately 27 kilocycles to a width of 270 kilocycles. As explained in the report, the expansion of the 27 megacycle band to a width of 270 kilocycles is based upon the recommendation of witnesses representing the medical service that a comparatively wide band of that width be made available for medical use. The Commission was assured by those witnesses that with this change its proposed allocations would prove satisfactory for medical purposes and that it would be practical to manufacture non-crystal controlled high frequency medical apparatus capable of satisfactorily operating within the width band in question with only a moderate increase in the initial cost of equipment and with no increase in maintenance expense. This is further discussed in the section of our report designated "Final Allocation for Industrial and Medical Use."

Your attention is also directed to the last page of the attached report which, in addition to summarizing the final allocation of frequencies for medical use, discusses the effect of that allocation on the operation of medical and industrial apparatus. As explained there, it is contemplated that equipment manufactured subsequent to May 25, 1945, the date of our report, will be required to be operated within assigned channels or in such a manner as to prevent interference to radiocommunication. Existing equipment will, in gen-

eral, be permitted to operate as before for a reasonable period of time to be designated later.

Very truly yours,
/s/ T. J. SLOWIE, Secretary

Enclosure

BASE HOSPITAL No. 64

The following details of the activities of General Hospital No. 64 which have been passed by the censor were sent by Col. C. J. Miangolarra, Chief of the Surgical Section and second in command of the Unit, under Col. T. H. Reagan. The Unit is sponsored by Louisiana State University School of Medicine.

After a little more than a year in camp at Fort Jackson, S. C., the Unit was sent overseas. Its first assignment was in North Africa near Terryville on the Tunis Road about fifteen miles from Mateur where it was set up in a tent hospital. The Army approved of this setup to such an extent that they used it as a pattern for similar units.

Dr. Miangolarra wrote that he was particularly proud of the surgical staff and felt that the excellent job it had done would be a credit to its sponsors and particularly to the Army. As an example of their activities during one twenty-four-hour period, he said that the Unit had handled 700 fresh casualties, none of whom spoke English, most of them being French and French colonials.

The second station was at Maddaloni which was near Leghorn. The Unit arrived at this location just seven days after the Germans had left. It was then moved to Ardenya where the Colonel facetiously remarked that the Germans were quite busy, shelling within a mile of the hospital and sending bombers over every night. Fortunately, there were never any direct hits on the hospital. At one time the hospital was caring for 2,800 patients.

While this is not official, it has been reported that the over-all mortality was in the neighborhood of one per cent.

CAPTAIN JACOBS HONORED

Captain Warren M. Jacobs, son of Dr. Adolph Jacobs, after completing his internship at Touro entered the Army. Recently Captain Jacobs was awarded the Bronze Star Medal. The citation reads as follows:

Captain Warren M. Jacobs, 0-426157, Medical Corps, United States Army, for meritorious service in connection with military operations against the enemy, from 11 February 1945 to 12 April 1945, in Germany. As Battalion Surgeon, Captain Jacobs carried out his duties in an exemplary manner despite the most adverse conditions. The speed and efficiency in which he handled casualties resulted in a remarkable record of achievement. His untiring effort throughout the period was an inspiration to his men. His ability, leadership, and devotion to duty reflect the highest credit upon Captain Jacobs and the armed forces of the United States.

By command of Major General Reinhardt:

H. Pengelly,
Lt. Col., A. G. D.,
Adjutant General.

NEWS ITEMS

The semi-annual refresher course in otolaryngology, which the University of Illinois conducts, will be held this year from September 24-September 29.

An illustrated lecture seminar integrating the patho-physiological reactions of the human to the environment and covering a wide range of medical problems of interest to the practicing physician will be conducted by Dr. William F. Petersen commencing Monday, September 17th, and extending through the 22nd. Sessions from 9:30 to 4:30 each day in the Conference Room of the Institute of Medicine, Chicago. Detailed information, program, registration applications, can be obtained from the Secretary, Institute of Medicine of Chicago, 86 E. Randolph Street (Crerar Library Building), Chicago. Enrollment limited.

Major Robert C. Kemp, of Baton Rouge, is now serving in an Army Medical Hospital in England.

The weekly bulletin from the Office of the Surgeon General announces the promotion of Daniel Joseph Fourrier, of Baton Rouge, from lieutenant colonel to the rank of colonel. Colonel Fourrier has been in the service since 1940.

Dr. W. H. Block is completing his fiftieth year in the practice of medicine. He attended at Baltimore on June 16th a reunion of the graduating class of 1895.

OFFICE SPACE AVAILABLE

Two full offices, with examining rooms, laboratory, dressing rooms on first floor. Second floor has operating room, kitchenette, eight hospital rooms with every hospital convenience. For further information communicate with Mrs. Winston Borum, 2221 Line Avenue, Shreveport.

BAPTIST HOSPITAL

The Clinical Staff of the Southern Baptist Hospital met at 8 p. m. May 22, 1945, Dr. Sam Hobson presiding.

The case of the month, "Acute Yellow Atrophy of the Liver," was discussed by Dr. R. W. Sharp, representing the Interne Corps.

Dr. H. R. Unsworth presented a case exhibiting psychiatric and neurologic findings due to advanced years and dietary deficiencies.

A motion picture entitled "Radium Treatment

of Cancer of the Cervix," filmed under the direction of Dr. Max Cutler, was shown.

Dr. Eugene Countiss presented "A Review of the Carcinoma of the Uterus Deaths at the Southern Baptist Hospital." His paper was discussed by Drs. J. T. Sanders and W. D. Beacham. Dr. P. M. Tiller gave a report of the deaths which included five cases of malignancy. There was one case of typhus fever and another of agranulocytosis.

The attendance at the meeting was excellent.

W. D. BEACHAM, Secretary

CHARITY HOSPITAL

The monthly meeting of the Medical Division of the Charity Hospital Visiting Staff was held on Tuesday, June 19, at 8 p. m. in the Hopsital Auditorium. The following program was presented: Torula Meningitis, by Drs. L. C. Hamilton and P. Thompson; The Salicylate Treatment of Rheumatic Fever in Children, by Drs. Rose Coleman and C. A. Stewart; a case presentation by Dr. S. Nelken.

OCHSNER CLINIC

A meeting of the Ochsner Clinic Staff members and fellows was held Friday, May 25, at 8:30 p. m. The program included two presentations, the first by Dr. Ralph Platou on congenital syphilis illustrated with numerous Kodachrome pictures. Dr. John C. Henthorne then discussed malignant cells in pleural effusions.

SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE—SOUTHERN SECTION

A meeting was held Friday, May 25, 1945, at 8 p. m. in the L. S. U. School of Medicine. The following program was presented:

Synaptic Connections in the Dorsal Root Ganglia. L. Matthew N. Bach (introduced by Dr. H. S. Mayerson), Department of Physiology, Tulane University School of Medicine.

Role of Antidromic Fibers in the Maintenance of Vascular Tone. L. Matthew N. Bach (introduced by Dr. H. S. Mayerson), Department of Physiology, Tulane University School of Medicine.

The Spatial Relationships of the Mean Electrocardiographic Axes. Richard Ashman, Department of Physiology, L. S. U. School of Medicine.

Pneumococcic Meningitis in a Newborn Infant with Recovery. Jesse Colley and Wallace Sako (Introduced by Dr. Richard Ashman), the Charity Hospital of Louisiana and the Department of Pediatrics, L. S. U. School of Medicine.

Studies on a Case of Congenital Absence of One Lung. Rena Crawford, John S. Ladue, and Chester A. Stewart (Introduced by Dr. Richard Ashman), Department of Pediatrics and Medicine, L. S. U. School of Medicine.

Corrosion Studies of the Blood Supply of the Human Kidney. George N. Ronstrom (introduced by Dr. A. G. Eaton), Department of Anatomy), Department of Anatomy, L. S. U. School of Medicine.

Plasma Chloride and Bicarbonate after Potassium Administration. Walter S. Wilde and Frederick P. Ferguson (Introduced by Dr. A. G. Eaton), Department of Physiology, L. S. U. School of Medicine.

INFECTIOUS DISEASES IN LOUISIANA

For the week ending May 5 the weekly morbidity report for the State of Louisiana contained a statement of the previous month concerning venereal diseases. During this month there were listed 1717 cases of gonorrhea, 1805 of syphilis, 30 of chancroid, 13 of granuloma inguinale and eight of lymphopathia venereum. For this particular week other reportable diseases included unclassified pneumonia 204 cases, cancer 161, pulmonary tuberculosis 138, malaria 53, pneumococcic pneumonia 46, measles 39, septic sore throat 19, mumps 16, whooping cough 11, and tuberculosis, other than pulmonary, ten. Three cases of poliomyelitis were reported this week from three different parishes in the state. Strange to say there were also reported four instances of smallpox. Thirteen of the patients with malaria contracted their disease outside of Continental United States. For the next week in May which ended on the twelfth, the following diseases were listed in numbers greater than ten: 53 cases of malaria, 26 of measles, 19 of pulmonary tuberculosis, 14 of unclassified pneumonia, and 16 of mumps. For the week which came to a close on May 19, listed in order of frequency, were the following diseases: pulmonary tuberculosis 32 cases, hookworm infection 27, measles 25, influenza 14, mumps and chickenpox 13 each, unclassified pneumonia 12 and 11 cases of malaria, all of which were contracted outside of the United States except one. One case of poliomyelitis was listed this week and one of epidemic encephalitis. There was apparently an epidemic of measles the week which terminated May 26 because there were reported 248 cases of this disease. Next in order of frequency there were reported 80 cases of pulmonary tuberculosis, 70 of malaria, 21 of unclassified pneumonia, 18 of septic sore throat, 16 of chickenpox and 12 of scarlet fever. Five cases of endemic typhus fever were reported this week and two cases of epidemic encephalitis. Of the 70 cases of malaria, only 18 were contracted outside of the United States. The report for the week which closed June 2 listed the venereal disease statistics for the previous four weeks. There were reported 2621 cases of gonorrhea, 1151 of syphilis, 26 of chancroid, 17 of granuloma inguinale and 12 of lymphopathia venereum. Other reportable diseases showed 176 cases of cancer, 129 of unclassified pneumonia, 76 of pulmonary tuberculosis, 41 of pneumococcic pneumonia, 27 of malaria, 22 of

measles and ten each of mumps and German measles.

HEALTH IN NEW ORLEANS

The Bureau of the Census, Department of Commerce, reported that for the week which ended May 12 there were exactly the same number of deaths in the City of New Orleans as there were in the previous week. The distribution of these deaths showed that 78 of the persons who died were white, 49 colored and 11 were children under one year of age; again this is the same number as in the previous week. For the week ending May 19 the number of deaths was almost identically the same as for the previous two weeks as 126 New Orleanians succumbed this week. Seventy-four of the deaths were in the white population, 52 in the non-white and eight were children under one year of age. For the week closing May 26 there was a sharp increase in the number of deaths, 155 people dying in New Orleans this week, 98 of whom were white, 57 colored, and ten children under one year of age. There were 20 less deaths for the week which closed June 2: 89 of these people who died were white and 46 were colored, with 11 children under one year of age. For the week which ended June 9 the 128 deaths were divided 86 white, 42 non-white and 13 in children under one year of age.

BRITISH HEALTH IN THE SIXTH YEAR OF WAR

The British Information Services supplies the following figures on British health for 1943.

In the first place, general health was better than it is usually in peace time. The number of births is the highest it has been since 1928; 30,000 more children being born than in 1942.

Deaths due to enemy action on the British Isles were 2367. These deaths, of course, occurred as a result of bombing.

There has been a continuous rise in the death rate from tuberculosis as well as a rise in the number of new cases. Venereal diseases have likewise shown a very considerable increase.

Occupational diseases present a new problem because of the many new substances used in the present day; plastics for example.

There is no evidence that there has been any increase in the serious cases of mental breakdown. There has been, however, a large number of eye injuries due to flying glass.

This very much summarized report of the British health in six years of war should be most encouraging to the British people. It is to be expected that tuberculosis and venereal diseases will increase during war but the excellent general health of the population apparently has been maintained in spite of stress of war and the probable food limitations.

DR. EDWARD F. BACON
(1875-1945)

Dr. Edward F. Bacon died at the age of seventy at Hotel Dieu, June 5. Dr. Bacon for many years was active in the Orleans Parish Medical Society and the Louisiana State Medical Society. He has had a long noteworthy career. He graduated from Tulane in 1904. Shortly after his graduation he was one of the doctors that helped combat the last yellow fever epidemic, acting as an assistant surgeon in the Public Health Service. During the World War he served in this country and abroad. For many years he was associated with the City and State Boards of Health. Generations of medical students will place Dr. Bacon as the brother of Mr. John A. Bacon, who was connected with the Tulane Medical School around forty years, and who though retired is still active and in excellent health.

DR. JOHN PRACHER
1883-1945

Dr. John Pracher, of Monroe, died March 30. He was clinical pathologist and bacteriologist for St. Francis Sanitarium from July, 1927, until his death. He was a member of the American Society of Clinical Pathologists, the Ouachita Parish Medical Society and the Louisiana State Medical Society.

One of Dr. Pracher's two children, a graduate of the L. S. U. School of Medicine, is a captain in the Medical Corps of the U. S. Army.

DR. LEWIS A. GOLDEN
1903-1945

The tragic death of Dr. Lewis A. Golden occurred in the Touro Infirmary on June 22 after a protracted illness. Dr. Golden, since his arrival in New Orleans nine years ago, had made a most enviable reputation for himself. He was looked upon as one of the outstanding psychiatrists in the community. He was a member of the staff of Touro Infirmary, Charity Hospital and the Ochsner Clinic. He was past president of the New Orleans Society of Neuropsychiatry and was a member of the Louisiana State Board of Institutions. He was consultant at the United States Marine Hospital, American Red Cross and the Child Guidance Center.

BOOK REVIEWS

We regret that the printers deleted in the June issue the last five lines of the review of "Patients Have Families" by Henry B. Richardson. These lines read: The conclusions are definite and the worker will be able to see the application of his efforts in the management of the case. The author is to be complimented on his excellent management of a difficult subject.

The author of this review is Dr. G. McHardy.

Forsdike's Textbook of Gynaecology: By J. H. Peel, M. A., B. M., B. CH. (Oxon), F. R. C. S., M. R. C. O. G., New York, Grune and Stratton, 1944, Pp. 440. Price, $5.75.

A textbook, well adopted for the medical student as well as for the general practitioner, has been considerably rearranged, the subject matter being presented in a most orderly fashion. New chapters have been inserted dealing particularly with the more modern ideas on the physiology of the female reproductive organs. The chapter on endocrinology is particularly clear. There has been appended a list of available hormone preparations used in this country and Great Britain. The text is well written, pleasant to read because of good print and paper, also contains excellent drawings and plates.

ADOLPH JACOBS, M. D.

Avitaminoses: By Walter H. Eddy, Ph. D., and Gilbert Dalldorf, M. D. Baltimore, Williams & Wilkins Company, 1944. Illus., 438 pp. Price $4.50.

The third edition of "The Avitaminoses" was indicated by the successful reception of the previous publications and further by the advance in knowledge on the subject during the three-year interval since the second edition. Demand for a third edition is a compliment to the authors and confirms the value of their efforts. Justification at this time for a re-editing and revision becomes apparent when one notes the extensive additions and changes in the text.

It is a remarkable text that should enjoy favorable comment. There are four hundred thirty-eight condensed pages with a minimal paper waste. When one realizes that this work evolved from "The Vitamine Manual" of 1929 which was written by the senior author and has developed to a well digested review of the enormous vitamin literature which requires a thirty-four page bibliography, an appreciation of the authors' efforts is forthcoming.

The title is misleading for the material covered embodies all aspects of vitamins, their chemistry, pathology, function, clinical aspects, the deficiencies at all levels, their relation to systemic and nutritional diseases, assay methods, diagnostic tests and the prophylaxis and therapeusis of deficiency.

The volume is divided into three parts. The first section of one hundred twenty-three pages is an evaluation of the vitamins, the second portion of two hundred twenty-eight pages covers the deficiency states and the third section of one hundred pages treats with the technical methods of vitamin assay.

It is an excellent, easily readable text, scholarly and complete. Despite necessary technicalities, the physiologic chemist and the clinician can read it with equal enjoyment as an authoritative coverage of our knowledge on a still rapidly evolving group of entities.

GORDON MCHARDY, M. D.

PUBLICATIONS RECEIVED

B-B Printing Company, Columbus, Ohio: Technical Methods for the Technician, by Anson Lee Brown, A. B., M. D.

The Blakiston Company, Philadelphia: Men Under Stress, by Roy R. Grinker, Lt. Col., M. C., and John P. Spiegel, Maj., M. C.

E. P. Dutton & Co., Inc., New York: Doctors at War, edited by Morris Fishbein, M. D.

The Foundation Press, Inc., Chicago: Law in Medical and Dental Practice, by Jack Neal Lott, Jr., and Robert Hanes Gray.

Garden City Publishing Co., Inc., Garden City, New York: Common Ailments of Man, edited by Morris Fishbein, M. D.

Grune and Stratton, Inc., New York: Textbook of Neuropathology, by Arthur Weil, M. D.

Paul B. Hoeber, Inc., New York and London: Fundamentals of Pharmacology, by Clinton H. Thienes, M. D., Ph. D.

Harvard University Press, Cambridge, Mass.: The Bacterial Cell, by Rene J. Dubos.

J. B. Lippincott Company, Philadelphia: Medical Licensure Examinations, by Harold Rypins, M. D., revised under the editorial direction of Walter L. Bierring, M. D., F. A. C. P., M. R. C. P. Edin. (Hon.).

W. B. Saunders Company, Philadelphia and London: A Manual of Surgical Anatomy, by Tom Jones and W. C. Shepard. Clinical Traumatic Surgery, by John J. Moorhead, B. S., M. D., D. Sc., F. A. C. S., (D. S. M.). Dietotherapy, Clinical Application of Modern Nutrition, edited by Michael G. Wohl, M. D. Penicillin and Other Antibiotic Agents, by Wallace E. Herrell, M. D., M. S., F. A. C. P. Preventive Medicine, by Mark F. Boyd, M. D., M. S., C. P. H. Physical Diagnosis, by Ralph H. Major, M. D. A Textbook of Ophthalmology, by Sanford R. Gifford, M. A., M. D., F. A. C. S.

Charles C. Thomas, Springfield, Illinois and Baltimore, Maryland: The Fundamentals of Electrocardiographic Interpretation, by J. Bailey Carter, M. D., F. A. C. P.